TASMANIA

REPORT OF THE ROYAL COMMISSIONER

(The Honourable Malcolm Peter Crisp
a Justice of the Supreme Court of Tasmania)

INTO

THE FLUORIDATION OF PUBLIC WATER SUPPLIES

HOBART 1968

1968 (No. 32)

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1968

D. E. WILKINSON, Government Printer, Tasmania.

34215

IN THE MATTER of Letters Patent under the Public Seal of the State of Tasmania dated the ninth day of December 1966

and

IN THE MATTER of the Evidence Act 1910.

To His Excellency SIR CHARLES HENRY GAIRDNER, Knight Commander of the Most Distinguished Order of Saint Michael and Saint George, Knight Commander of the Royal Victorian Order, Knight Commander of the Most Excellent Order of the British Empire, Companion of the Most Honourable Order of the Bath, Lieutenant-General on the Retired List of the Army, Governor in and over the State of Tasmania and its Dependencies in the Commonwealth of Australia.

SIR,

I have the honour to present a Report on the subject of Fluoridation of Public Water Supplies, a matter entrusted to me as a Royal Commissioner by Letters Patent under the Public Seal of the State dated the ninth day of December 1966.

I have the honour to be Sir,

Your obedient servant,

(Sgd.) PETER CRISP, *Commissioner*

DATED at Hobart, in Tasmania, this eleventh day of July 1968.

(Sgd.) R. J. HICKMAN, *Secretary*

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PART A—TERMS OF REFERENCE

1. On 9 December 1966 by Letters Patent under the Public Seal of the State, I was appointed a Royal Commissioner to inquire into and report upon the desirability or otherwise of the fluoridation of the public water supplies of this State. My terms of reference were as follows:—

'(1) *Whether in Tasmania material benefit to dental health can reasonably be expected from the addition of fluoride to water supplied to the public having regard to the present state of scientific knowledge and to experience with respect to water supplies containing fluorides, whether naturally or by addition, in other States of the Commonwealth and elsewhere.*

'(2) *Whether any detriment to health or other public disadvantage may result from the addition of such quantity of fluoride to water supplied to the public as, with fluoride naturally present, will not exceed in the aggregate one part per million.*

If your recommendation and report should favour the addition of fluoride to water supplied to the public then *to report upon the following further matters, that is to say—*

'(3) *Whether it is practicable and desirable to leave the decision whether or not to add fluoride to water supplied to the public to local or other public authorities responsible for such supply and if not to whom should such decision be entrusted?*

'(4) *Whether there are any practicable methods of ensuring an adequate daily intake of fluoride by those whom it is sought to benefit other than by the addition of fluoride to water supplied to the public and, in particular, whether the supply of fluoride tablets to individual members of the public could be a satisfactory substitute.*

'(5) *What would be the cost of adding fluoride to water supplied to the public and whether, as a public charge, it would be justified in the public interest.*

'(6) *Whether the existing statute law of this State and in particular the statutory scheme embodied in section* 61 *of the Public Health Act* 1962 *is adequate in its scope and effect to allow of the implementation of any decision to add fluoride to water supplied to the public and if not what amendments are necessary or desirable?*

And *generally to inquire into and report upon such other questions coming to your notice in the course of your inquiries into the foregoing as you consider should be investigated in connection therewith.'*.

2. The time for reporting in respect thereof was subsequently extended to the thirty-first day of July 1968 by Letters Patent dated the twenty-third day of May 1967, and the first day of November 1967.

PART B—GENERAL

CHAPTER 1. SITTINGS, EVIDENCE AND PROCEDURE

Section (a)—Sittings

3. The Commission opened at Hobart on 20 December 1966. It commenced taking evidence on 15 March 1967 and completed its sittings on 21 March 1968. The Commission sat on sixty-six sitting days mainly in Hobart, but shortly at Scottsdale, Beaconsfield and Ulverstone as well. It was subject to many interruptions, twice due to the illness of myself but also, and more importantly, due to the necessity which arose in the latter part of 1967 for me to return to full-time judicial duty during the absence of the Chief Justice from the State. During this time the proceedings of the Commission were of necessity entirely suspended.

4. In addition to such easily recognisable matters the nature of the inquiry necessitated by the terms of reference made it imperative to go outside this State for evidence and submissions. Fluoridation is a controversy of world status. There are both advocates and opponents of fluoride whose names are internationally known and no inquiry could hope to achieve anything which did not seek their assistance. It is said that probably no current topic in the field of public health has been so intensively studied and debated. Apart from the difficulty of selecting and studying a comprehensive bibliography on a subject where the total published items were, as long ago as 1963, estimated[1] to include 16,000 scientific papers (and their number is being added to daily), it was necessary to invite experts in various fields of chemistry, dentistry, medicine and public health from other parts of Australia to come to Hobart to give evidence, as well as to invite submissions by correspondence from known advocates and opponents of fluoridation in other parts of the world, notably the United States of America.

5. The effect of these matters upon the course of the Commission's inquiry has not generally been appreciated. The powers of a Royal Commission to subpoena witnesses and compel their attendance do not extend beyond the boundaries of the State. By far the bulk of the expert oral evidence necessary to be taken in the course of these proceedings has resulted from negotiated attendances in the course of which the sittings of the Commission have had to be fitted to the convenience of busy men whose willingness to attend was not always matched by their ability to adjust rigid academic schedules and other professional appointments to the Commission's needs. This is by way of explanation, not by way of complaint. As Commissioner I have nothing but gratitude to all (in both camps) who have so freely sought to assist me and so generously given of their knowledge; not only those who, from practically every State in Australia, attended as witnesses, but those too whose physical attendance distance made impracticable but who sought to assist the Commission with detailed submissions and extensive documentary material.

6. Some interstate witnesses were inclined to question the emphasis which the Commission placed upon personal appearance where at all practicable. The attitude of the Commission, which was communicated in writing to those who questioned it, was one that will be readily appreciated by those acquainted with forensic values, but perhaps not so readily by others. Witnesses were told, that the Commission, while it could not insist upon a personal appearance, nevertheless preferred it for the obvious reason that in a subject of such differing views and personalities, a witness who appeared in person to present his thesis and to defend it against inquiry and cross-examination would, as a result of such testing, be of more assistance to the Commission than otherwise. However, nothing has been excluded and all submissions whether backed by personal appearance or not have been read and considered.

7. Originally the public was informed of the Commission's intention to take evidence and invited to make submissions by advertisement, but this was supplemented by express invitation in those cases where the person concerned was known to have expressed views either for or against fluoridation which it was thought the Commission should investigate. I also made it a point to go wherever in this State it was learnt that a witness resided who wished to give evidence and whose attendance in Hobart could not otherwise be arranged. I deliberately chose to proceed upon the basis that, so far as this State was concerned, no witness should be excluded who wished to appear, no matter what his professional or academic status.

Section (b)—Witnesses and Exhibits

8. The names of the witnesses who did appear to give evidence and of the persons and organisations who, unable to appear, favoured the Commission with submissions (in some cases extending to hundreds of pages) are given in Appendices 'A' and 'B'.

9. Perusal of these appendices will give but little indication of the scope of the inquiry. I have heard evidence from professors who needed blackboard and chalk to reproduce the symbols of their speciality as well as from the unlettered who spoke perhaps less eloquently but with equal conviction of their fears and sometimes their prejudices. I have listened to doctors, dentists, chemists, biochemists, pharmacists, analysts, engineers, as well as ordinary citizens, politicians, councillors, farmers and housewives. The evidence has ranged from the prehistoric Tasmanian aboriginal to the Eskimo, from Tristan da Cunha to the Punjab, from the laboratory marvel of a fluoride-free diet fed to germ-free rats to the tuckshops of Australian school children. From Fascism to Communism, though strangely the trail ended with Zionism, from ions to aeons, volcanoes to kettles, from null point potentiometry to the social habits of the edentulous, from acne to allergy, baldness to beer, tinea to dandruff and in the range of medicaments, drugs and chemical compounds, from thalidomide to Glauber's salts. A major preoccupation has been the teeth and bones of humans, rats, dogs, cattle, sheep and hamsters, ground, pulverised, sectioned, ashed, stained and dissolved.

10. There are few of the ordinary pathological conditions of both medicine and dentistry that have not been mentioned and indeed, some of the extraordinary ones as well. The traditional seven stages of life have had to be extended to cover the prenatal and the preschool, as well as the aged, the pregnant, the undernourished and the alcoholic. With this material I have endeavoured to cope but it would be too much to hope in the examination which the evidence has obliged me to make of a subject so variegated in content, so diverse in the scientific disciplines that it called in aid, that, surfeited with such strange fare, symptoms of dyspepsia could altogether be suppressed. If, despite care, there are errors of technical expression, and I fear in such unaccustomed fields inevitably solecisms will be found, I seek to protect myself by saying that this Report is intended for laymen, to be read by laymen and not as a technical treatise.

11. Appendix 'C'* is a list of the exhibits received in evidence. Apart from a few dental specimens probably not numbering more than ten they consisted in the main of monographs and articles extracted from the scientific and technical literature of the world, including in some cases languages other than English. They ranged from pages to volumes.

12. This was in truth a battle of 'the papers' learned and otherwise. Some hundreds were produced to the Commission while inquiries pursued led to still more. Not all witnesses were capable of maintaining a scientific detachment in their choice. Indeed, with such a monumental mass of published material to draw upon, it would be difficult to do so and some witnesses reflected in their selection of exhibits more bias than discrimination. However, nothing was excluded and all has been read, and the major task has been to digest and assess the comparative worth of all this diverse material, whether scientific, polemical, political, pseudo scientific or frank nonsense (and there was some in all those categories). At times it seemed beyond the capacity of one man to do so but, that apart, the sheer bulk of the material submitted has necessarily involved a drastic curtailment of the normal procedure of discussing in the course of judgment the evidence in detail. To reflect in this Report all the arguments used; to seek to detail even in synoptic form all the evidence and to explain all the steps by which a conclusion has been reached would entail many volumes and more months perhaps than I have weeks.

13. It follows that some witnesses will not find in the text of my Report explicit reference to their evidence or submissions. Some may be disappointed, some may even be relieved. It is to be remembered that not many of the witnesses spoke from direct personal knowledge of the matters to which the Commission's attention was being directed. Most spoke from conclusions formed and convictions gained as a result of their reading and not in consequence of personal research. In such circumstances it is but natural that the focus of attention should be the argument or the article and not necessarily the person who advanced it.

14. This was particularly true of those who opposed fluoridation, even among those whose names have been publicly prominent in this controversy. I would except at once Dr Sutton and Dr Waldbott and probably Professor Steyn all of whom may fairly claim to have done original work. Their work and their evidence or submission is discussed on an individual basis in its

* This Appendix is not reprinted. In the main the exhibits received were the articles, submissions and other documents, specific references to which are given at the end of each section of the Report.

proper place. Others like Sir Arthur Amies lent weight to the argument by their attainments in the general field of science or dentistry but not by any original work of their own in the subject being discussed.

15. Others again like Professor Polya and Dr Exner (particularly the former) have been in the past concerned more with the polemics of the matter than the science[2]. This is not a field into which I have thought it necessary to inquire except where compelled. There have been many excesses and some quite irresponsible things said and done in the course of public controversy even in this State. I have been astonished and in fact dismayed by the vituperation and the unbridled arguments *ad hominem* employed by men who not only claim but in fact possess high scientific attainments. But it will not advance my inquiry to seek to rake over the undignified details of the matters to which I refer. Happily before me I think it can be said, while echoes of such matters intruded at times, there was a genuine attempt to keep things on a rational plane. I therefore intend to avoid, as far as I can, digressing into questions of personalities, though, as I have said, some attention will have to be given to the individual attitudes of some professional or scientific witnesses and others whose writings and viewpoints have been brought prominently before the public mind.

16. However, let me say now, I do not seek or wish to be construed as seeking to impugn the good faith of anyone who either gave evidence before me or favoured me with a submission. Controversy is never pleasant and I was impressed by the sincerity and desire to help manifested even by those who may have regretted being put in the position where old unhappy far-off things and battles long ago might have to be refought. I am grateful to all witnesses, not only for what they contributed to my understanding but for permitting the proceedings of the Commission to be conducted without heat or rancour no matter what the temperature this controversy may have attained in the past. Insofar as I have to refer to some aspects of those things, I do so with some regret, and only so far as I judge it to be necessary to evaluate present evidence.

17. The evidence of the lay witnesses was not all of equal weight. This was inevitable. Some were misguided even to the point of paranoia but others reflected the uncertainties of the intelligent citizen confronted with social measures which he interprets as encroaching on traditional liberties. I am grateful to two in particular, the late Mr Geo. Gray, M.H.R. and Mr T. W. Davenport of Ulverstone. Both were dedicated opponents of fluoridation yet managed to retain a considerable degree of objectivity and were of great assistance in impartially furthering inquiries by the Commission. With them I would also bracket, though to a lesser extent, Mr L. J. Kausman of Melbourne. Though I have not felt able to adopt their viewpoint, they all commanded the Commission's respect.

Section (c)—Procedure

18. One further matter should be mentioned as the method of the Commission's functioning. Surprise has no part in an inquiry of this sort and from the beginning a policy was formulated and adhered to that proofs of opposing witnesses were to be exchanged as far as practicable so that they could be studied before evidence was given, not only by counsel but by other witnesses of equal status who might hold opposing views. The consent of witnesses was of course necessary, but I am not aware of a single instance where any objection was raised, and I am confident that this procedure did much to promote a full and informed exchange of views. In addition, where the writings and views of overseas scientists and other authorities were taken into account and matter challenging their views was advanced, if it seemed necessary to the cause of an objective inquiry, it was sought to refer the challenging statements, whether they went to substance or to credit merely, to the persons challenged and their answer invited. This was done in some cases through representatives of the associations and interests aligned with the authority whose answer was sought and in other cases by correspondence directly with officers of the Commission. Most responded without rancour and answered in the spirit in which the inquiry was made. I regret, however, in one instance the Commission was embarrassed by a threat to use in another country, as a basis for legal proceedings, the information which the Commission had furnished to permit the person concerned to reply. I have no opinion as to the likely success or otherwise of the proceedings threatened but apart from this the Commission has been fortunate in the co-operation it has received from all concerned, whatever their country, their qualifications and their views.

Section (d)—Previous Inquiries and Reports

19. Fluoridation has been the subject of a number of previous inquiries and reports. They are listed and their findings summarised in Appendix 'D'. I refer to two in particular.

20. The South African Commission of Inquiry reported on 12 May 1966. Its Report however was not available in this State till late in 1967 when the evidence of the Commission was all but complete. It was a Report made following a public inquiry lasting two years by a highly expert Commission of six members, five of whom were qualified academically and professionally in medicine, dentistry or some other branch of science. In relation to the medical and dental aspects of fluoridation this has seemed to me the most valuable Report of all as the combined experience and knowledge of the Commission enabled it to speak with a technical confidence which reflects the subjective appraisal of skilled assessors. I have used it extensively as will appear from the text, not only as an authoritative reference, but as a touchstone against which to test many of my own tentative conclusions.

21. During the course of the Commission I learnt that the World Health Organisation which had already in 1957 adopted a Report of an Expert Committee favouring fluoridation had resolved to organise the preparation of a monograph on 'The Metabolism of Fluorides and their Utilisation in Medicine, Dentistry and Public Health'. Professor Yngve Ericsson of Sweden was chosen and has acted as a special consultant in the planning and preparation of the monograph and twenty-eight scientists of international standing in this field have contributed to its contents.

22. Unfortunately, though I have been generously permitted access to the draft of this Monograph, it had not at the time of writing my Report, progressed sufficiently far towards adoption and publication to enable me to refer to it as I would wish as being either in final form or as representing the views of the World Health Authority. Until finally settled and adopted it would, of course, be improper so to represent it. However, in those cases where the views of contributors to the Monograph have seemed to me to possess special significance either because they refer to work as yet unpublished or because of the international repute of the contributors, I have with the consent of the World Health Authority referred to the text of his contribution, employing the following formula:—

> 'A B (name) "Title of Chapter" (1967)—Unpublished Contribution to provisional text of a Monograph on "Fluorides and Human Health" in course of preparation by World Health Organisation'.

Section (e)—Legal Decisions

23. Appendix 'E' lists a number of legal judgments referred to in evidence. There are many others in which legal questions arising in fluoridation disputes have been considered[3]. Most have been concerned with questions of statutory interpretation as to whether proposals to fluoridate were 'within power' under the terms of legislation relating to municipal water supplies. This question has, generally speaking, been put to rest by an affirmative decision of the Judicial Committee of the Privy Council[4].

24. Another case in which the merits of fluoridation, as distinct from its legality, were debated and discussed at length, is the decision of Kenny J. of the Irish Supreme Court in the case of *Ryan* v. *Attorney-General*[5]. The decision is cited extensively in the literature and is interesting because many of the world-wide figures (e.g., Professor Steyn, Drs Waldbott and Exner) in the fluoridation controversy appeared and gave evidence. For this reason I have found it useful on occasion to refer to the transcript which has been available to me.

25. However, apart from their general interest, I have not, of course, regarded any of those cases as having any force as precedents (in the legal sense) in an inquiry of this nature.

Section (f)—Bibliography

26. Most Reports of this nature would include a select bibliography of works consulted. In a subject of this magnitude this is impossible on any satisfactory basis. A reference to the List of Exhibits (Appendix 'C') will indicate the difficulty. I have confined Appendix 'F' to major treatises and monographs of a general nature but both sides are represented in the works listed. References of a more specific nature will be given in the chapters in which the matters to which they relate are discussed.

REFERENCES

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CHAPTER 2. APPEARANCES BY COUNSEL

27. When the proceedings of the Commission were being planned it was foreseen that there would be a clear line of division between those who favoured and those who opposed fluoridation. Some of the contest was scientific, much was statistical and there were peripheral areas of conflict ranging from the political to the frankly emotional. In such circumstances of conflict the role of counsel assisting the Commission, at all times difficult, becomes almost impossible. His primary responsibility is to the Commission to advise in the collection of evidence, to appraise its value and to present it. But he has a secondary role that is just as important and that is, on behalf of the Commission, to test the evidence by cross-examination as vigorously as he feels the circumstances warrant. Such cross-examination is best conducted by counsel who have not previously identified themselves with the witness concerned. This identification is impossible to avoid if counsel has also had the responsibility of taking instructions from the witness and briefing his evidence. Furthermore, the witness is apt to be puzzled and even to resent any attempts at cross-examination by counsel with whom he has previously met and discussed his evidence on a basis of intimacy.

28. Initially it was sought to free counsel's hands by appointing a solicitor to carry out the initial interviews and the briefing, but it soon became apparent that owing to the technical nature of the questions involved this was not enough and counsel assisting the Commission had of necessity himself to interview witnesses. While counsel himself was always clear as to his role, witnesses were not and the danger quickly, in fact immediately, became apparent that witnesses, even partisan ones, identified themselves with counsel assisting the Commission and when called and examined were perplexed and sometimes dismayed when their reception became more searching and critical than they had anticipated.

29. In short, in a matter of this sort, where there are two sides strongly opposed, it is too much to expect one counsel both to present evidence and as well to test it by cross-examination. If he confines himself to presenting the evidence, then it is left to the Commissioner himself to step into the arena and conduct his own cross-examination. By so doing he imperils his objectivity and the confidence which the witnesses must feel in a tribunal of this sort to ensure its successful functioning.

30. For these reasons it became apparent very early in the proceedings that unless some steps were taken to have separate representation of the two opposing bodies of opinion, the Commission would be greatly handicapped. I am grateful to the Minister for Health for making it possible for Mr Cosgrove of counsel to represent the Anti Water Fluoridation Council of Australia and New Zealand and Mr Baker of counsel to appear for the Australian Dental Association. Later Mr Cosgrove was assisted by Mr Lyons of counsel for the Tasmanian Pure Water Association. The assistance of these gentlemen in the presentation of opposing views added depth and content to the debate and has in my mind made inapplicable to the proceedings of this Commission the criticisms made in other parts of the world of similar Commissions and Boards of Inquiry that the views of opponents of fluoridation had not been adequately presented because of the lack of 'reasonable assistance towards the cost of gathering evidence and presenting the case'.

CHAPTER 3. ACKNOWLEDGMENTS

31. The task of gathering, collating and presenting this world-wide material has, of course, fallen mainly on the officers of the Commission. They were Mr R. C. Jennings of counsel and Mr D. A. Burton, solicitor assisting the Commission and Mr. R. J. Hickman, my associate, who acted as its secretary, assisted in the Departmental aspects by Mr E. C. Coleman of the Public Health Department. I cannot speak too highly of their indefatigable efforts. The scope of their inquiries can to some extent be judged by the number and quality of the submissions received and the list of exhibits and other appendices attached to this Report, but they are but the visible portion of the iceberg.

32. I am also greatly in the debt of the Officers of the State and University Libraries and in particular to Miss Margaret Ramsey of the Tasmanian State Library who guided my first approaches to the literature on the subject and to Miss Virginia Logan of the same institution who has subsequently sought and obtained needed references from libraries and publishers in Australia and elsewhere. To them and those other librarians, unknown but understanding, whose forebearance in the matter of non-returned inter-library loans has helped me so much, I also express my gratitude.

33. Above all I am grateful for the competent assistance of my typist, Mrs J. H. Swete, who has coped not only with my drafts but displayed admirable patience with hundreds of references to technical monographs and serials in an unfamiliar medium. Lastly too, I express my thanks to my attendant and tipstaff, Mr M. W. Sims, who has coped not only with me but with many things, as he always does, efficiently and with good humour.

PART C—ANSWERS TO QUESTIONS PUT TO COMMISSION AND SOME GENERAL CONSIDERATIONS AFFECTING THEM

CHAPTER 4

Section (a)—Questions and Short Answers

34. My short answers to the questions raised by the terms of the Commission are as follows:—

(1) Q. *Whether in Tasmania material benefit to dental health can reasonably be expected from the addition of fluoride to water supplied to the public having regard to the present state of scientific knowledge and to experience with respect to water supplies containing fluorides, whether naturally or by addition, in other States of the Commonwealth and elsewhere.*

A. Yes, substantial and material benefit. (Part 'F')

(2) Q. *Whether any detriment to health or other public disadvantage may result from the addition of such quantity of fluoride to water supplied to the public as, with fluoride naturally present, will not exceed in the aggregate one part per million.*

A. No, subject to the following minor qualifications:—

(i) There is a risk of dental fluorosis occurring in some children. The number affected will not exceed 10% of the child population and may be less. The degree will be 'mild' (probably about 2%) and the remainder either 'very mild' or 'questionable'. There is no reason to fear that it will in any case be disfiguring or even noticeable except to a clinical observer. In any case, over the child population as a whole it will be more than counter-balanced by the improved shape and appearance of the dentition and by an even greater reduction in mottling and staining of teeth from other causes. (Part 'G', Chapter 21)

(ii) There is a possibility that some individuals will exhibit a hypersensitivity not shared by the rest of the population to fluoridated water. The possibility is extremely remote, so remote that I cannot quantify it statistically. The fact that such individuals do exist has not been clearly demonstrated but the possibility that they may exist cannot be dismissed. If they do exist the reaction to be expected is that of an abnormal toxic response to low dosages, not an allergic reaction in the strict sense. Hence in relation to water fluoridated at 1 ppm the reaction would be mild, easily relieved and prevented. (Part 'H', Chapter 24)

These qualifications I regard as minor. I believe the community advantage lies overwhelmingly on the side of fluoridation. My recommendation and report should therefore be taken to favour it. In these circumstances I am required to report upon the following further matters:—

(3) Q. *Whether it is practicable and desirable to leave the decision whether or not to add fluoride to water supplied to the public to local or other public authorities responsible for such supply and if not to whom should such decision be entrusted?*

A. No. The decision whether or not to fluoridate the communal water supplies of the State should be made by Parliament. (Part 'K', Chapter 51)

(4) Q. *Whether there are any practicable methods of ensuring an adequate daily intake of fluoride by those whom it is sought to benefit other than by the addition of fluoride to water supplied to the public, and in particular, whether the supply of fluoride tablets to individual members of the public could be a satisfactory substitute.*

A. There are other means of administering a fluoride supplement to the diet of the community, but in point of efficacy, as public health measures, they fall well below fluoridation of communal water supplies. In particular the supply of fluoride tablets to individual members of the public or their children would not be a satisfactory substitute as a measure of public health. (Part 'L', Chapter 52)

(5) Q. *What would be the cost of adding fluoride to water supplied to the public and whether, as a public charge, it would be justified in the public interest.*

A. No clear-cut answer can be given to the first part of this question as there are too many variables. Fluoridation would be most economically effected in conjunction with regional schemes of water treatment and supply. If this were the pattern the greater proportion of the State's population could be given fluoridated water at an annual cost of approximately 20 cents per head of population served. However this is an estimate only and it is a matter of individual cost analysis for each project. Without co-operation on a regional basis, this figure could be substantially exceeded in the case of small separate local schemes particularly those supplying less than 3,000 consumers. Subject to the above I consider that the expenditure likely to be incurred would be justified in the public interest in the light of the benefits to health which may be anticipated. (Part 'M', Chapter 53)

(6) Q. *Whether the existing statute law of this State and in particular the statutory scheme embodied in section* 61 *of the Public Health Act* 1962 *is adequate in its scope and effect to allow of the implementation of any decision to add fluoride to water supplied to the public and if not what amendments are necessary or desirable?*

A. No. The existing statute law is not adequate in scope and effect to allow of the implementation of a decision to fluoridate. If such a decision be made by Parliament, then I indicate in Chapter 44 *post* my views as to the manner in which such a decision should be implemented and the authorities to whom it should be entrusted. (Part 'N', Chapter 54)

Section (b)—General Comments

35. I endeavour to enlarge those answers and to indicate reasons in what follows. But the field is large. It will be noted that in the first question the issue is stated in terms of dental health; in the second it is broadened to include physical health in all its aspects as well as matters of public advantage or disadvantage generally. In later questions, political, engineering and legal problems are involved.

36. A simple summation of them all would be to ask 'Is there a problem of public health to be met, is fluoridation an answer, is it efficient, is it safe and are there other answers?' This is the approach which underlies the remainder of this Report. But so that my affirmative answer to these questions may be understood, it is necessary first to discuss simply the basic chemical and physiological processes that are involved. I am required to have regard 'to the present state of scientific knowledge' in relation to the subject. I have endeavoured as a layman to inform myself of the rudiments, but I make no claim to expertise or to scientific training or qualifications of any sort. I stress, no doubt unnecessarily, that the Commission itself has conducted no research, no chemical experiments, no scientific tests. It is the effect of what others have established that I seek to convey and I seek to do so in terms which will enlighten and not confuse the uninitiated. As a familiar, if difficult judicial task, I have endeavoured first to assimilate, then to assess the evidence proffered and in reporting to state as a layman for other laymen my understanding of its worth and effect.

37. In fulfilling such a task there is no room for absolutes. The ultimate boundaries of science and knowledge can never be discovered with certainty, the speed with which in any age they may be thought to recede is only matched by the vastness of the vistas that lie beyond. Absolute truth is known neither to the scientist nor the judge, all that can be hoped for is to indicate the measure of persuasion that the evidence available induces. I attempt no more.

PART D—BASIC FLUORINE CHEMISTRY

CHAPTER 5. DEFINITION OF FLUORIDATION

38. Basically fluoridation is the addition of fluoride to the diet with a view to lessening both the severity and the incidence of dental caries. Though other dietary forms are known, it has come to be associated predominantly with the addition of fluoride to communal water supplies. Proponents of the measure recognise an optimum dosage which, in temperate climates, may be taken to approximate in volume one part of fluoride to a million parts of water, or, as a percentage, 0.0001%. In this sense, as indicating an adjustment of the fluoride content of drinking water to recommended optimum levels, the term is used in this Report.

CHAPTER 6. ELEMENTARY FLUORINE CHEMISTRY

39. Science, and in particular chemistry, subdivides the physical world into ultimate substances or elements. In very simple terms, and ignoring such things as isotopes which nobody has suggested are directly relevant to this investigation, these are particles of matter which can exist or be demonstrated as pure, i.e., as unmixed with other substances and as possessing an individual structure. The ultimate or smallest possible particle of such an unmixed substance or element is known as an atom. An atom can be distinguished from the atoms of other elements by factors of measurement. These factors involve concepts of atomic weight and number which it is unnecessary to describe, save that the concept of number has reference to a further division of the atomic substance into particles of energy of which the electron is the only one relevant to this inquiry. But that is to descend the scale. Ascending, the next smallest particle of matter is the molecule. These are mixed or complex substances involving a combination of two or more elemental atoms in chemical bond or union. As with an atom, the term is used to describe the smallest possible quantity of such a union of elemental particles.

40. Fluorine is an element, one of the 103 ultimate substances known to science. In the language of chemistry it reacts violently with other elements, that is, it so readily combines with other substances to form compounds, that in a free, i.e., an unmixed state, it is not found in nature. It can be produced in a pure elemental form in a laboratory, but even then the simple atom has but a transient existence combining instantly to form a fluorine molecule of two atoms. The molecular species thus formed is a gas with toxic properties.

41. As a non-metallic gas, fluorine falls into the elemental sub-group of substances known as halogens. The others are chlorine, iodine and bromine. From time to time in the fluoridation controversy, suggestions have been made that fluorine derivatives or fluorides may compete with, displace or display similar physiological characteristics to other halogens, notably iodine in relation to the thyroid gland. Such arguments, generally speaking, rest on analogy only and not on demonstration. Reference will be made to the subject later but for the moment it is sufficient to say that, considered as a halogen, fluorine differs materially from the other halogens in its valency, its atomic weight and its electronic configuration. I have found no support in the scientific literature for any argument as to the physiological behaviour of fluorine and its compounds based on any supposed analogy between fluorine and other halogens[1,2].

42. Fluorine then exists in nature in combination with other elements. An important consequence arises from the fact that chemically there is no necessary relation between the intrinsic toxicity or noxious qualities of an element and those of any of its compounds. They vary widely and each chemical compound must from this aspect be considered as a separate entity. Thus, to argue that elemental fluorine is a poisonous gas therefore any chemical combination involving the same element must possess the same toxic properties, is chemically fallacious.

43. In the literature raising some of the early objections to fluoridation this essential pharmacological fact does not seem always to have been appreciated. This is not a peculiarity of fluorine alone but is a characteristic shared with other elements and their compounds. Indeed wide differences in physical characteristics may even be found between different or allotropic forms of the same element where no compound is involved at all, e.g., carbon, phosphorus and sulphur.

44. An example of a compound with physiological properties quite different from those of its component elements and which is frequently cited is common salt formed by a combination of the elements sodium and chlorine. There are others which are even more striking, such as compounds of phosphorus and potassium. The toxicity of elemental phosphorus is well known, but

many of the phosphates which it forms in combination with other elements are vital to human life processes. The converse may also occur. Neither carbon nor oxygen would in the ordinary sense be regarded as toxic, but in combination as carbon monoxide, they can be lethal. All this is very elementary but the importance in this controversy of getting away from the type of argument which depends on some woolly analogy with elemental fluorine cannot be overstated.

45. Chemical compounds are conventionally divided into two great groups, the organic in which carbon is one of the combining elements, and the inorganic in which it is not present. I am not primarily concerned with organic compounds of fluorine except to rebut certain suggestions which have been advanced. They are extremely rare in nature, being unknown in the animal or mineral kingdoms and being confined to two forms associated with poisonous plants found in Africa and with the gidyea tree (*Acacia Georginae*) of the Australian continent. It is not, incidentally, found in Tasmania.

46. The poisonous principle of these plants, which in the case of the Australian tree is fluoracetic acid, is the same as that of the synthetic Compound 1080 (sodium fluoroacetate) which has been mentioned in the evidence. It has been extensively studied. Sir Rudolph Peters[3, 4] has demonstrated conclusively that the toxic effects of the fluoracetate molecule are due to the effect of the molecule as a whole, that is, it depends on the carbon fluorine bond remaining intact. Putting it another way, in these only known examples of an ability to synthesise by biological means an organic fluoride compound the nature of the poisoning induced by the complex organic molecule thus produced has no connection with fluorine *per se* but is due to the whole molecule and not to its fluorine content.

47. Artificially produced organic compounds of fluorine for industrial and therapeutic purposes now number some thousands and their number is constantly being added to. The general stability of the carbon fluorine bond means that they do not constitute a suitable vehicle for fluoridation and they have never been considered for the purpose. They are therefore strictly irrelevant to this inquiry. However, a suggestion made in evidence that dietary fluoride may be supplemented by fluoride released in the metabolism of drugs containing organic fluorine compounds to the extent that levels of intake may be significantly raised will be considered later. (See Part I, Chapter 42.)

48. A suggestion[5, 6] which, were it true, would give rise to far more serious implications, was that organic substances in the diet may combine with fluoride in water (introduced in the course of fluoridation) so as to form organic fluorine compounds of unknown and uncontrollable toxic potential. This I am satisfied is a chemical impossibility. Reduced to a homely example, the addition of fluoridated water to vinegar (acetic acid) will not, I understand clearly, produce fluoroacetic acid, which as we have seen is the active principle of Compound 1080 and is a very toxic substance. Common sense would suggest that it would indeed be surprising if it did. However the evidence of Dr O'Donnell, Associate Professor of Inorganic Chemistry at the University of Melbourne, a specialist in fluorine chemistry, was quite specific 'there is no known aqueous route to the preparation of fluoro-organic compounds' and it has been specifically denied by Sir Rudolph Peters, whose rank as a world authority on the subject of fluroacetic acid can hardly be questioned.

49. This same suggestion was considered exhaustively by the New Zealand Commission at p. 63 of their Report. Having learnt that it had been linked with the name of Sir Rudolph Peters, the Commission investigated and reported in the following terms:—

> '203. The fact that this is an absurd misrepresentation of the researches of Sir Rudolph Peters, formerly Professor of Biochemistry at Oxford, was explained by Mr Francon Williams, Assistant Director of the Dental Division, Department of Health. He referred us to a letter written by Sir Rudolph Peters and published in the *Lancet*, 26 January 1957, as follows:—
>
> "FLUORIDATION: A MISQUOTATION
>
> Sir,
>
> In a pamphlet from California entitled *Fluodidation Unmasked*, the following statement about myself appears:—
>
> Professor R. A. Peters, Biochemist of Oxford, England, says:
>
> 'Fluoroacetic acid prevents the body from breaking down the citric acid created when sugar is burned into simpler compounds. Retention of waste citric acid acts as a violent poison on heart and nervous system. Fluoroacetic acid is used by African natives to murder their enemies. What will happen to the person drinking fluoridated water when he uses vinegar?'

I do not know upon what writing of mine this is supposed to be based, but I think it as well to point out that it gives entirely the wrong impression. Inorganic fluoride acts completely differently in the body from fluoroacetic acid, in which the carbon is linked firmly to the fluorine. The suggestion in the last sentence that fluoride and acetic acid will combine easily to form fluoroacetic acid is very wide of the truth. It is in fact quite difficult to synthesise fluoroacetic acid, and certainly there is no evidence that it can happen in the body. Furthermore, acetic acid (vinegar), if given early enough, is an antidote to fluoroacetic acid poisoning in some animals.

R. A. PETERS,
A.R.C. Institute of Animal Pathology
(Biochemistry Department),
Babraham, Cambridge.".

204. In September 1956, Dr G. N. Davies, Otago University Dental School, wrote to Sir Rudolph Peters drawing his attention to the fact that this assertion had appeared in this anti-fluoridation pamphlet. In his reply to Dr Davies dated 1 October 1956, Sir Rudolph Peters stated, *inter alia*:—

" Thank you for your letter of 19 September. I am astonished at this statement attributed to me, and should be most grateful to have the address of A. Wilkinson so that I can ask him for a reference. I have never said that ' retention of waste citric acid is the cause of poisoning '. It is not fluoroacetic acid which is used by African natives in the Sierra Leone region. The last sentence in the quotation is meaningless.".

205. This evidence is quite conclusive. The statement contained in the pamphlet which indicates that the extract is in the words of Professor R. A. Peters is untrue and the extract itself is a garbled misrepresentation of the facts.'.

50. The related suggestion made by Sir Arthur Amies, which I only reproduce because of its source, that there was a possibility that other organic fluorides may be formed in fluoridated water by a combination of the fluoride ion and other organic substances already in the water and that this possibility had not yet been investigated[7] was answered by Professor O'Donnell in the following terms:—

'I have provided during my evidence in Hobart, documentary evidence that there is no known aqueous route to the preparation of fluoro-organic compounds. If this is so, why would any chemist embark on the type of study which Sir Arthur Amies proposes? There are no chemical principles which would suggest even vaguely that such reactions would occur. Naturally no investigation will be carried out. It would be a complete waste of time and would not be justifiable research.'.

51. In the extensive world literature on the subject of fluorides and fluoridation I am not aware nor has Sir Arthur drawn my attention to anything which may be taken as supporting the suggestion that he makes either in theory or in fact. As such it remains a speculation, apparently uninformed, which gains no support from any of the evidence and must be rejected.

52. In contrast to organic forms, inorganic forms of fluorine are common in nature. Just how common will be discussed in the next chapter. But before going on to discuss some general matters relating to them, there is a matter of terminology to be settled. The terms ' fluorine ' and ' fluoride ' are used indiscriminantly in the literature and they can confuse the observant but uninformed reader. When used as a substantive it is clear that ' fluorine ' properly refers to the elemental atom F or to the molecule F_2. Fluoride, on the other hand, is generally used to denote the fluorine component of an inorganic compound containing fluorine. Examples are sodium fluoride, NaF, calcium fluoride, CaF_2, and sodium silicofluoride, Na_2SiF_6 to name three which are of importance to this inquiry.

53. The term ' fluoride ' also reflects the fact that in most inorganic combinations or salts the method of bonding between fluorine and the other element or elements involved is ionic, i.e,, there has been an exchange of orbital electrons. This means that the fluorine atom has been changed in the process of ionization by the loss of one electron, and the derivative term ' fluoride ' is used to denote its ionized form. The ion is symbolised at F^-. The process also involves a change in electrical charge between the atom and its ionized form. On the other hand, in organic compounds (and in some few inorganic forms) the method of bonding is different, there is an electron sharing and the same reasoning may not apply. I shall throughout this Report use the term fluorine to indicate the element F or the molecule F_2, while fluoride will be used to indicate derivative forms or salts such as calcium fluoride CaF_2 or the fluoride ion F^-.

54. The implications of this ionic process are fundamental to an understanding of fluoridation. When in combination with other elements as an inorganic salt, the elemental fluorine atom has been structurally changed and its physical properties altered. When dissolved in water the ionized particles of the molecule of the compound dissociate as a result of the process of hydration or hydrolysis or a combination of both. It is not necessary to describe these processes. The important thing in simple language is that when the elemental particles of the dissolved molecule dissociate, they do not regain their elemental form but continue to retain their ionized form. We have seen that there are physical differences of structure and electrical charge between the two forms. It is a consequence of such differences that the atom of an element and the ion of the same element have differing chemical characteristics and behave differently from one another. This means that in any question of toxicity or noxious principles in the present context it is the fluoride ion that must be considered and not elemental fluorine.

55. Just as all atoms are alike by virtue of their elemental nature, so all ions of the same substance are alike irrespective of their source, and have exactly similar properties and characteristics. The source of a fluoride ion, i.e., the compound from which it was derived, is immaterial in any assessment of its effect, in fact a chemist can only postulate and cannot demonstrate by any known system of specific analysis the particular source of fluoride ion in solution. This is stressed because of the contention frequently advanced that there is some intrinsic difference between fluorides naturally occurring in water and those artificially introduced. Such a contention which appears to gain support from some vague theories of nutrition, sometimes described as naturism, has no scientific support and indeed is described by one prominent opponent of fluoridation, who on this point may at least claim to speak with authority, as a sort of mystical concept[8] and as flowing 'from ignorance of elementary chemistry'[9].

56. To summarise what has been said, the properties of elemental and molecular fluorine are not relevant to this inquiry, nor are the organic compounds occurring in nature. Of the chemically manufactured organic compounds of the element, while the carbon fluorine bond is exceptionally stable and unlikely to be broken in the course of human metabolism, the remote possibility that dietary fluoride may be supplemented by fluoride metabolically released from such compounds, will be examined later in connection with the only field in which this effect is likely to be of any significance, namely the therapeutic administration of complex organic drugs in which fluorine atoms play a part.

57. Inorganically combined fluorides or salts are the only significant natural source of the element available physiologically to man. In such salts, the element is present in ionized form, and when such salts pass into solution the ion is dissociated from the other ion or ions in which it may previously have been combined but retains its ionic form. As the fluoride ion it has individual characteristics and properties which are different from the elemental atom or its molecular species F_2.

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CHAPTER 7. DISTRIBUTION AND ABUNDANCE OF FLUORIDES

58. In ionized or inorganic form fluorine is so common in nature that it ranks seventeenth among the elements in order of abundance. As a constituent of the earth's crust it has an average concentration ranging from 400 to 800 ppm[1]. Its distribution is universal, and it is certainly a significant, if not an essential, element in the human environment. The impossibility of postulating a completely free fluoride diet is generally conceded and it is a normal constituent of the human body. It is found in appreciable strength in sea-water (at levels generally estimated to exceed 1 ppm), in the atmosphere, in rain-water and in varying concentrations in most, if not all, potable waters.

59. It has been said and I accept it as indubitable, that 'advances and improvements in analytical techniques have led to the verification of its presence in varying amounts in many natural materials, and today it is doubtful, when the size of the sample is adequate, that air, water, vegetation, or food materials absolutely free from fluoride can be found'[2].

60. In a primary natural form fluoride is found in a wide variety of mineral forms in rocks and soils. It occurs most commonly as fluorite or fluor-spar (calcium fluoride CaF_2) but other important sources are cryolite (a mixed compound containing sodium and aluminium fluorides) and rock phosphates (in the form of apatites). The occurrence of these salts is not confined to deposits of fluoride containing minerals in which the fluoride levels are so high that the deposits are of commercial value, but they occur almost universally in rocks and soils of the earth's crust, in varying proportions, ranging from tens to thousands of parts per million[3].

61. Though rocks and soils may be considered as the primary source of fluoride in man's environment, other derivative sources are not without interest and importance. Sea-water contains significant quantities, levels having been variously recorded from 0.8 to 1.4 ppm[3], though some higher levels are claimed[4]. Fluoride is also widely distributed in the atmosphere, its commonest sources being the dusts of fluoride-containing soils, the gaseous contents of industrial wastes and the smoke of coal fires. In the presence of heavy industrial pollution, the concentration can be significant[5], otherwise the levels are greatly variable ranging from 0.9 to 18 parts per billion (American usage) (i.e., 0.0009 to 0.018 ppm) being reported for a number of American and British cities[2].

62. These sources may be reflected in the fluoride concentration found in rain-water or precipitation. In areas of heavy industrial pollution, a reported concentration of 3.4 ppm is cited by Waldbott[6] but on the literature this would appear to be clearly exceptional and the general agreement is that in normal circumstances the amount contributed by rain-water is small[3] unless augmented by evaporation or contact with fluoriferous material. One analysis recorded by Reid and Martin of the rain-water contents of a galvanised tank in a country centre in N.S.W. gave 0.05 ppm[7].

63. However, while these constitute environmental sources of fluoride, the main ingested sources are of course food and drink, supplemented to a small degree by fluoride inhaled from the atmosphere. It seems clear that fluoride is present in most, if not all, foodstuffs. The difficulties of preparing, even with the most refined techniques, a totally fluoride free diet are generally believed to be insuperable, though attempts have been made in order to study the results in laboratory animals. Certainly it is true to say that no normal human diet can be postulated free from fluoride. One associated difficulty should be mentioned. Accurate methods of quantitative analysis of some of the micro quantities involved have only been evolved in the last two or three decades, and in the case of animal soft tissues, probably only in the last decade. New and improved methods of water analysis are now available as described by Professor O'Donnell in evidence, but some of them are still novel. This means that a great deal of the earlier literature dealing with the quantitative analysis of trace amounts of fluoride (say below 0.05 ppm) has to be treated with caution and this is particularly so where, as is common in the literature referred to, the analytical methods used are not described.

64. The subject of the fluoride content of foodstuffs has been studied by McClure[8] in particular and his figures for 130 types of foodstuffs are universally quoted. The length of the article cited forbids its reproduction in full. It is particularly interesting in that he includes data designed to show the effect of adding varying concentrations of fluoride to the diet or the nutrient water of the animals and plants studied. A table prepared by Muhler[9] and summarising some of his figures is attached for information as Appendix 'G'. No comprehensive figures related to Australian foodstuffs specifically are available, but no significant difference is to be anticipated. Cholak[2] reviewing world literature arrived at the figures shown in Table I, but he points out, as do other writers[9], that the higher figures in the ranges given, as a rule are exceptional and in some instances are based on a single finding which may not be representative.

65. With three important exceptions, tea, beer and sea-food, the fluoride content of foodstuffs is generally low, though some common items are in the medium range, such as white bread and cereals about 1 ppm[10] and cheese and chicken with about 1.5 ppm[11]. Some forms of salt may have very high values, but the amount ingested has to be related to climate, dietary habits and the amount used.

66. By far the most important source of dietary fluoride is drinking water. Rain-water has already been mentioned. It may gather atmospheric fluoride in falling or acquire it in the hydrologic cycle by mechanical transfer from sea-water[12]. Unless it evaporates or runs off, it may on penetrating the soil come into contact with fluoriferous material and the further amount of fluoride accumulated will depend on the geological structure of the area.

67. As a general rule, surface waters though invariably containing some[13] are low in fluoride, the levels being below 1 ppm and in most cases considerably below. There are exceptions[2]. Subsoil or underground water will have greater opportunity for contacting fluoriferous material and depending on the geology of the area may acquire much higher fluoride concentrations. While exceptional values have been obtained in some South African lakes (2800 ppm) and in mineral springs up to 100 ppm, such waters would not be regarded as potable. The subject has been extensively studied. In a comprehensive review Cholak[2] summarised the results obtained in various parts of the world and his summary is reproduced in Table II. It is doubtful whether any inference can be drawn from those figures save that of diversity. Strangely—in the light of this controversy—many of the mineral springs long esteemed for their therapeutic properties are highly fluoridated, e.g., Carlsbad.[2] Though in South Africa water from certain bore holes and wells containing fluoride in concentrations up to 53.0 ppm have been used by native populations[14], a figure of 14 ppm such as has been reported in some United States communities, would now be regarded as dangerous to health and in an advanced community steps would be taken either to obtain an alternative supply or to reduce it.

TABLE I

	Ppm Fresh	Ppm Dry
Meats	0.01–7.7	3.8–7.7
Fish	<0.10–24	–84.5
Citrus fruits	0.04–0.36	1.4–2.2
Non-citrus fruits	0.02–1.32	0.42–12
Cereal and cereal products	<0.10–20	0.0–64
Vegetables and tubers	0.10–3.0	0.0–28.3
Miscellaneous material:		
Tea	3.2–400	–1900
Wine	0.0–6.34	....
Beer	0.15–0.86	....
Milk	0.04–0.55	....
Salt (table)	0.02–115.1	....
Bone meal (edible)	246–770	....
Coca Cola	0.07	....
Eggs	0.00–2.05	....
Butter	0.4–1.50	....
Sugar	0.10–0.32	....
Baking powders	<0.10–220	....
Cheese	0.13–1.62	....
Coffee	0.2–1.6	....

Taken from Cholak[2]

68. Australian figures are, generally speaking, low as are New Zealand, and Tasmanian figures are perhaps even lower. In 1946 Reid and Martin[7] determined the naturally occurring fluoride content of 93 samples of potable waters taken from N.S.W. and Tasmania. It was estimated that they represented the common water supplies of 85% of the population of N.S.W. and nearly 50% of that of Tasmania. Of the total 80 were surface waters and 74 of these had a fluoride content of 0.2 ppm or less. Of the 13 underground waters, nine contained fluoride to the extent of 0.5 ppm, the highest level observed being 1 ppm.

69. No fluoride at all was detected in five of the six Tasmanian samples collected covering the major centres of population in the State and only 0.1 ppm in the remaining one. Later figures supplied by the Tasmanian Government analyst give a slightly but not a significantly different picture. They are reproduced as Table III. They represent an up-to-date summation of the analyses obtained in his Department over the last ten years, and while the samples taken would not by any means be sufficient to establish average yields, they are, taken in conjunction with earlier but perhaps not quite as reliable figures, sufficient to give the overall picture.

70. This table establishes clearly that Tasmanian communal water supplies are relatively low in natural fluoride. In not a single instance have samples taken exceeded 0.05 ppm and the average was estimated by the analyst at between 0.03 and 0.04, some being recorded as the merest trace at 0.01 ppm., i.e., one hundredth of a millionth part. Such an average would seem to be significantly lower than those obtained for Australian and New Zealand communities generally as appears from studies reviewed by Bell and Ludwig in 1967[8].

71. I think it would only be time-wasting to embark upon and to reproduce further detailed comparisons with other parts of the world when the conclusion and the inference to be gained is so clear. Tasmanian drinking waters are notably low in their natural content of fluoride by comparison with other parts of the world. If fluoridation to an optimum level of 1 ppm is necessary in the interests of dental health, then they may be regarded as gravely deficient, if it is not then their deficiency is a matter of no consequence.

TABLE II

THE OCCURRENCE OF FLUORIDE IN VARIOUS WATERS OF THE EARTH

Location	Parts per Million Average	Range
North America:		
The U.S.A.—		
Alabama	....	0–6.8
Arizona	....	<1.0–33.5
Arkansas	0.10	0.0–3.0
California	....	0–7
Colorado	....	0–5
Connecticut	0.12	0–0.4
Delaware	0.09	0–0.2
Florida	0.21	0–2.5
Georgia	0.20	0.0–2.0
Hawaii	....	0–2.22
Idaho	....	0.3–5.0
Illinois	0.49	0–4.0
Indiana	0.25	0–2.0
Iowa	....	0–2.8
Kansas	....	0–10.0
Kentucky	0.5	0–3.9
Louisana	....	0–6.0
Maine	0.11	0–0.4
Maryland	0.04	0–5
Massachusetts	0.12	0–8
Michigan	0.22	0–2.0
Minnesota	0.16	0–3.2
Mississippi	....	0–6.0
Missouri	....	0–5.0
Montana		No records
Nebraska	....	0–2
Nevada	....	0–5
New Hampshire	0.11	0–0.5
New Jersey	0.08	0–2.1
New Mexico	....	0–12.0
New York	0.09	0–1.3
North Carolina	0.13	0–4.2
North Dakota	....	0–7.5
Ohio	0.17	0–3.0
Oklahoma	....	0–2.4
Oregon	0.03	0–0.5
Pennsylvania	0.06	0–2.0
Rhode Island	0.08	0–0.8
South Carolina	0.48	0–7.0
South Dakota	....	0–1.2
Tennessee	....	0–2.0
Texas	....	0–7.2
Utah	....	0–5
Vermont	0.02	0–0.1
Virginia	0.12	0–7.5
Washington	....	0–5.0
West Virginia	0.14	0–0.6
Wisconsin	....	0–2.8
Wyoming	....	0.1–1.2
Canada	....	0–1.2
Mexico	....	0.1–17.5
Cuba	....	0–0.4
South America:		
Argentina	....	0–1.6
Brazil	....	0–0.60
Ecuador	....	0–6.3
Peru	....	0–1.4
Europe:		
Austria	....	0.38–0.8
Belgium	....	0–1.68
Cyprus	....	0–3.6
Czechoslovakia	....	0–28.00
England	....	0–4.5
Finland	0.57	....
France	....	0–7
Germany	....	0–4.90
Hungary	....	0–5.14
Ireland	....	0–0.2
Italy and Sicily	....	0–7.1
Luxembourg	....	0.02–1.23
Netherlands	....	0–2
Norway	....	0.01–0.07
Poland	....	0–1.1
Portugal	....	–22.8*
Russia	....	0–4.6
Spain	....	0.0–6.3
Sweden	....	0.0–1.12
Switzerland	....	0.01–1.42
Yugoslavia	....	0–4.16
Africa:		
South Africa	....	Trace–53.0
East Africa (Kenya)	....	0–<30
Tanganyika	....	Trace–95
Asia:		
China	....	0–13.0
India	....	0–>3.0
Japan	....	0.02–20.0
Korea	....	0.8–10.0
Israel	....	0.3–1.50
Iran	....	0.0–<1.0
Australia	....	0.0–13.5
New Guinea	....	0.0–<0.55
Papua	....	0.0–<0.55
New Zealand	....	0–0.9

Taken from Cholak[2]

* Mineral waters only

TABLE III

FLUORIDE CONTENT OF TASMANIAN COMMUNAL WATER SUPPLIES AS DETERMINED BY GOVERNMENT ANALYST, HOBART

Town	Source	Fluoride (as F) (parts per million)
Beaconsfield	South Esk River	<0.05
Bellerive	Derwent River Southern Regional)	<0.03
Blythe, Howth and Heybridge	Lings Creek	0.03
Bothwell	River Clyde	0.03
Branxholm	Black Creek	0.05
Bridport	Brid River	0.05
Brighton	Derwent River (Southern Regional)	<0.03
Burnie	Pet River	0.01
Campania	Derwent River (Southern Regional)	<0.03
Campbell Town	Elizabeth River	<0.05
Castle Forbes Bay	Castle Forbes Rivulet	0.01
Cygnet	Agnes Rivulet	<0.05
Devonport	Forth River	<0.05
Dover	Esperance River	0.01
Fingal	South Esk River and Fingal Rivulet	<0.05

Town	Source	Fluoride (as F) (parts per million)
Franklin	Fleurtys Creek and Swamp Creek	0.01
Geeveston	Kermandie River and Scotts Rivulet	<0.05
George Town	North Esk River (North Esk Regional)	0.05
Glenorchy	Derwent River, Lake Fenton and Knights Creek	<0.03
Gormanston	Creeks (names unknown)	0.02
Hamilton	Clyde River	0.04
Hobart	Mt. Wellington catchment, Derwent River and Lake Fenton	0.03, 0.04, & 0.03
Huonville	Huon River and Mountain River	<0.05
Kempton	Derwent River (Southern Regional)	0.03
Latrobe	Mersey River	0.03
Launceston	St Patricks River	0.03
Legerwood	Ringarooma River	0.05
Maydena	Junee River	0.04
Midway Point	Derwent River (Southern Regional)	<0.03
New Norfolk	Derwent River, Upper Lachlan, Illabrook and Lake Fenton	<0.04
Oatlands	Blackmans River	<0.05
Penguin	Penguin Creek	0.04
Queenstown	Hunter Creek and Roaring Meg Creek	0.02 & 0.03
Railton	Whitewater Creek and Mersey River	0.02 & 0.03
Richmond	Derwent River (Southern Regional)	<0.03
Ringarooma	Vineys Creek	0.04
Rosebery	Stitt River, Mountain Creek and Rosebery Creek	0.03
Ross	Macquarie River	0.03
St Leonards	North Esk River (North Esk Regional)	0.05
Scottsdale	Great Forester River	<0.05
Sheffield	Lake No Where Else and Snake Rivulet	0.03
Smithton	Deep Creek and Duck River	0.01 & 0.03
Somerset	Cam River	<0.03
Sorell	Derwent River (Southern Regional)	<0.03
Sulphur Creek	Sulphur Creek	0.03
Swansea	Swan River and Meredith River	0.01
Ulverstone	Gawler River	<0.05
Wynyard	Camp Creek and Inglis River	<0.03
Yolla	Johnsons Creek	<0.03
Zeehan	Parting Creek	0.03

(< = less than)

Note by Analyst: It is estimated that the above results cover the water supplies for more than 75% of Tasmania's population and are considered to be fully representative due to the widely dispersed sources.

72. The source of fluoride in naturally fluoridated waters is fluoriferous material with which the water comes in contact. It would seem to be generally true, and I accept it, that the deeper the source the higher the concentration of fluoride that may be expected, though in the last resort it is the geology of the area and not the depth that is decisive: It would also seem to be clear that the major source of the fluoride ion in such naturally fluoridated waters is calcium fluoride but it is not correct, as was asserted before me, that this is the invariable source. Thus Chalmers Clarke[15] has demonstrated in South West Lincolnshire, a naturally occurring soft water with a high fluoride content derived from sodium fluoride. This occurs as a result of a natural base-exchange process of water softening whereby weak solutions of relatively insoluble calcium fluoride are converted into soluble sodium fluoride. A similar composition is claimed for some of the other naturally fluoridated waters in England. Other naturally fluoridated soft waters are reported in Illinois[16], Colorado Springs and in South Africa[17]. In Illinois at least, the mechanism would seem to be similar, if not the same, as in Lincolnshire. I have no data for the others.

73. However, despite these interesting examples and ignoring some other examples of water analysis (including some reported for Queensland) in which fluoride concentrations are reported or expressed by an analysts' convention as sodium fluoride without meaning to bear any relation to the source of the ion, the general but not invariable rule would appear to be that naturally fluoridated waters are hard, i.e., they generally contain calcium and magnesium ions in high concentrations. This is claimed to be a factor of some significance by some opponents of fluoride and will be considered in due course.

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CHAPTER 8. FLUORIDE AS A CONSTITUENT OF THE HUMAN BODY

Section (a)—As a Normal Constituent

74. It is clear that fluoride is a normal constituent of the human body. Whether it is an essential constituent is a different but related question which will be discussed in the next section. Difficulties of accurate analysis, particularly of the soft tissues and the blood, have thrown doubt on some of the early work in this field but it is now considered that dependable and adequate techniques have been available at least for the last decade[1] and a great body of experimental and analytical data has been accumulated both in relation to human beings and animals.

75. According to Armstrong in the work cited[1] the application of modern analytical techniques 'always reveals fluoride to be present in mammalian body-fluids and tissues, and further, that particularly large amounts occur in calcified tissues'. Such results have been demonstrated in areas where the natural fluoride available from communal water has fallen well below the optimum of 1 ppm postulated by the fluoridationists.

76. Fluoride is retained in different parts of the body in different concentrations. In human blood plasma and soft tissues generally a level of 0.14 to 0.19 ppm has been demonstrated and the maintenance of this level is remarkably constant. On the other hand, in bones, teeth and other calcified tissues, much higher levels are found and these increase with age and intake. Fluoride is deposited in calculi (stones) and in other centres of calcification such as occur ectopically in the placenta, the aorta and other organs. The placenta does permit the passage of fluoride, though it may regulate the quantity that passes to the foetus. These are subjects which will have

to be discussed in more detail later, my purpose in mentioning them here is to stress the universality of fluoride, both in nature and in the human body, as a preface to the next section which discusses the question whether fluoride should be considered an essential element.

Section (b)—As an Essential Constituent

77. The concept of trace elements is one that is generally understood. Most Australians are familiar with the notion that minute quantities of certain elemental substances such as zinc, copper, cobalt, etc., are essential to life or health and that deprivation is followed by a deficiency disease. The question has been discussed in the literature at some length whether fluorine should be regarded in a similar light. Its determination, even affirmatively, would still leave unresolved the further question of its quantitative administration, so that it will not resolve the larger questions here being considered, but if it is properly so regarded, then the idea of an optimum dosage may be the more readily accepted.

78. There is an initial difficulty of terminology; of definition. Is a trace element one that is essential to the maintenance of life or can it be one that is essential to health merely, i.e., is it a question of mortality or morbidity? I note that the opponents of fluoridation would by definition exclude the latter sense. According to Waldbott[2] 'an essential trace element to be so designated must be proven to be required for existence of life'. On the basis of that definition he claims that 'nowhere in the scientific literature has fluoride been proven necessary for maintaining life'. We may accept this as correct, i.e., no experiments have been conducted to my knowledge which purport to show that deprivation of fluoride, either totally or in degree, is followed by the death of the subject. But morbidity—disease—is a different matter, and the evidence that in order to function as a healthy organism the body needs and cannot do without small but significant amounts of fluoride preponderates. If I err in what after all may be mainly a semantic matter in thinking that this warrants classification as a trace element, then at least I do so with Sir Macfarlane Burnet whom I feel is reasonable company to keep[3].

79. At times the argument tended to shift ground to a related but semantically different question, 'Is fluoride an essential nutrient?', thus raising the question of what is a 'nutrient' as well as when it may be said to be essential. As to this I take leave to quote and adopt what has been said about a similar phrase 'dietary essential' in the same context[4].

> 'The words have quite different meanings to different people, depending very often on which field of investigation the research worker belongs to. Many nutritionists would hold that a dietary essential is a nutrient that cannot be synthesized by the organism in sufficient quantities to maintain life, but must be ingested in some regular fashion during the entire life span of an organism if premature death is to be avoided. Microbiologists might state that a dietary essential is a food or food factor that is necessary to maintain the normal flora of nonpathological organisms within the individual. Physiologists have defined dietary essentials as foods that provide for the normal maintenance of specific functions of the organism. By such a definition, a particular food factor may be recognized as essential for a specific function of an organism; if the body is deprived of it, the organism as a whole will not die, but the particular function will be impaired.'

80. These semantic difficulties can serve to confuse. I only pursue the matter because of the seeming importance attached to it by the opponents of fluoridation and because it contributes to what I call (for lack of a better term) the philosophical objection to the same process. I will assume—anticipating reasons to be elaborated later—that fluoride has a caries preventive effect. But I will also assume that caries may be prevented in other ways so that it cannot be said to be an essential preventative in the sense in which 'essential' is used in this context, i.e., one in the absence of which there is disease. I would also grant that no presumption of benefit arises from its universality in the chemical composition of the human body, its presence being equally consistent with environmental contamination as with any teleological argument. Nevertheless it must be true to say with Kehoe, as quoted in the *Lancet*[5].

> '. . . fluoride is . . . part of the internal environment of the human organism. It cannot, in this relationship, be, in itself, harmful.'

81. A review (1960) by Gordonoff and Minder[6] would seem to be the most balanced summary of the case made by the opponents of fluoridation. However, their case is that the physiological function of fluoride is not yet established, not that it has none.

'Fluorine also is a regular constituent of the inanimate and animate worlds. It is found in water, in soil, and in the living organism, more particularly in bones and teeth. However, several generations of rats can be raised on a diet entirely free of fluorine without the least adverse effect of any vital function. As we shall discuss below, anticariogenic activity has been attributed to fluorine. In contradistinction, however, to what is observed in the truly "essential" elements, it is never, even according to the most optimistic accounts, 100% effective. Most authors credit fluorine with a 30 to 40% success; indeed this value has almost become a "magic number", since all investigators attempt to evaluate their results statistically in comparison with it. It cannot so far be stated, with confidence, that fluorine is an element essential to life.'

82. A reference is not given but the statement 'several generations of rats can be raised on a diet entirely free of fluorine' would in 1960 be taken to refer to the work of Maurer and Day (1957)[7]. The statement 'entirely free of flourine' is not literally accurate but sufficiently so perhaps to indicate that the authors claimed to have maintained the rats in question on a diet estimated to contain no more than 0.007 ppm of 'utilizable fluorine'.

83. It is clear that a diet 'entirely free' from fluoride is an impossibility[8] so ubiquitous is fluoride in the atmosphere, in food, and in fluids. If the statement be further taken to mean that rats were raised without the possibility of fluoride entering into physiological processes, it could be misleading, because in fact in the relevant experiments fluoride in significant quantities was found in the soft tissues and skeletal structures of all the experimental animals, even the unborn young.

84. Maurer and Day in the study referred to (in conflict with earlier and similar studies by McClendon and Gershon-Cohen (1954)[9]) found that rats maintained on a minimal fluoride diet did not show any significant difference in growth and well-being as against other rats maintained on the same diet supplemented by water containing 2 ppm F. Their conclusion from these studies was that fluoride was non-essential in nutrition at least for the rat and that 'its value in the body is apparently limited to the promotion of resistance to dental caries'.

85. These studies and other early ones on the same lines have been examined in detail by Muhler (1959)[4]. He claims that in all the experiments except those of McClendon and Gershon-Cohen 'measurable quantities of fluorine were used' and thus the effect of an entirely free fluoride diet has not been tested. It is however worth noting that in the McClendon and Gershon-Cohen experiments, in which by hydroponic means strenuous efforts were made to reduce the fluoride dietary content to a very low level, experimental animals uniformly developed caries and other dental defects. It was also claimed that growth and reproduction were affected. Muhler's own conclusion (in 1959) was that:—

'It can be stated with certainty that there is strong suggestive evidence from the studies of McClendon and Gershon-Cohen, as well as from human studies in natural and artificial fluoride communal fluoride areas, that fluorine is essential for dental health. This in itself should classify fluorine as an essential element. Also, if fluorine is essential for the 'normal' formation of enamel and dentin, perhaps it plays a more fundamental role in the calcification process in general. This area of investigation needs much further study, and it is premature to classify fluorine as 'non-essential' for human nutrition as a result of limited rat studies.'

86. However, since the work referred to, new factors have entered the controversy. Professor Storey directed attention to recent work which he said 'demonstrated the rationale by which fluorine has a function in body physiology'. This was the work of Taves and Neuman (1964)[10] who have shown for the first time that fluoride at levels normally found in human serum can enhance calcification in the presence of physiological concentrations of magnesium if the product of the calcium and phosphate concentrations is lower than normal. Their findings are taken to suggest that serum concentrations of fluoride can act as a governor or chemostat minimising the effect of fluctuations in tissue phosphate levels[11]. Another study by Newesely (1961)[12] repeated by Perdok (1963)[13] is said to show that the formation of bone, of which the mineral component has an apatite structure, would not be possible under biological conditions without the presence of fluoride at physiological levels. The South African Commission[14] refers to this study as demonstrating that fluoride is essential for the development of the foetal skeleton. Yet other studies referred to by Professor Storey may be taken increasingly to indicate the importance of the role of fluoride in stabilizing crystal structure by enhancing the structural integrity and stability of the calcified tissue[13]. These effects in relation to bone are now thought to retard senile rarefaction and osteoporosis in man[20].

87. Professor Davies[15] of Queensland has directed attention to the increasing evidence that with fluoride, as with other trace elements, experiments which neglect inter-actions with other elements in what may be a complex and not a simple effect, can produce inconsistent results, i.e., that physiological reactions may be the result of a synergism or of interlocking mechanisms of different elements and that studies which in the past have sought to establish a role for a single element without also taking into account its relationship with other elemental factors in the environment may mislead. He refers to Nikiforuk (1961)[16] suggesting that there are important ratios between fluoride and other salts which, if not maintained, might give unexpected results if attention were given to the presence or absence of one element only. See also the work of Malthus *et al* (1964)[17] and Taves and Neuman already referred to[10].

88. These matters, while awaiting further study, do a great deal to enhance the importance of fluoride in the physical environment. They suggest that when all these matters have been fully evaluated a far more complex and perhaps a dominant role may be found for fluoride that goes beyond the mere strengthening or improvement of the crystalline structure of bones and teeth as the tendency has been to claim in the past.

89. In summary it is obvious that fluoride has at least a beneficial role in the human physiology. The evidence goes beyond a mere presumption of benefit arising from its universal presence in the human body. Its cariostatic role can be taken to be definitely established but as caries may be prevented in other ways, e.g., by limiting cariogenic factors in the diet, it cannot on that account be said to be essential. Its further role in the human physiology awaits final verification, but it certainly cannot be said that there is no evidence pointing to its essential nature. On the contrary, I am of the opinion the preponderance of evidence points to it as possessing a role in the initiation and perfection of calcification processes which is vital to skeletal and dental health[18].

90. I do not think it necessary to explore the semantic differences of the opposing sides any further, except to add this. Whether fluoride is or is not a nutrient is to me a matter of terminology not deserving of the emotional mystique with which it has at times been enshrouded; sometimes deepened by the addition of the word 'essential'. In this regard I would refer to the findings of the highly expert South African Commission[19]:—

> 'Since the body is incapable of synthesising fluoride and since there is excellent evidence to indicate that dental decay can be diminished by taking optimal amounts of fluoride, a good case could be made for the official recognition of fluoride as an essential nutrient. This was the opinion expressed by Dr A. R. P. Walker during the course of his evidence before the Commission. There is, furthermore, good recent evidence to indicate that fluoride is necessary for the formation of apatite structures under biological conditions. (Para. 129.) While we do not feel competent to define fluoride as an essential nutrient, we are none the less agreed that the case for so doing is sufficiently strong to invalidate the argument originally stated and we have accordingly rejected it.'

91. I agree with the conclusion stated in the last sentence. The case for or against fluoridation should depend on its intrinsic qualities, not on labels.

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CHAPTER 9. FLUORIDE METABOLISM

92. In this chapter any attempt comprehensively to outline the present state of scientific knowledge or theory of the subject discussed, is expressly disclaimed. Full reviews exist, in particular by the South African Commission (1966) and in the provisional text of the Monograph in course of preparation by the World Health Organization referred to in Chapter 1, § 21 of this Report. The following treatises may also be consulted—'Handbook of Experimental Pharmacology' Vol. XX; 'Pharmacology of Fluorides' Part I, Ch. II by F. A. Smith (1966); 'Mineral Metabolism', Vol. II; 'The Elements', Part A, Ch. 24; 'Fluoride' by H. C. Hodge (1964); 'Fluorine Chemistry' (J. H. Simons, ed.), Vol. IV by H. C. Hodge and F. A. Smith (1965). However in order that the rest of this Report may be intelligible to the reader who has not access to these and other sources in the world literature, some attempt is made to summarise pertinent aspects.

Section (a)—Fluoride Absorption—General

93. Absorption of dietary fluoride (from both food and drink) depends in the first place on the solubility of the particular compound ingested. No account is here taken of organic compounds. Whatever relevance they possess to this matter is discussed under the later head of 'Objections to Fluoridation'. Inorganic compounds can be classified as—

(*a*) Soluble, e.g. sodium fluoride or sodium silico fluoride;
(*b*) Relatively insoluble, e.g. calcium fluoride and fluoroapatite (as in bone-meal); or
(*c*) Inert, e.g. potassium fluoroborate.

94. In the physiologically inert compounds of the last group the stability of the compound is so great that no fluoride ions are released. They are therefore of no significance to a study of fluoridation[1].

95. Insoluble fluoride if taken in solid form may be only partially absorbed within the body and the balance will be directly excreted in the faeces. If it is already in solution, as we have seen in our discussion of ionization, the source ceases to be material and the ion is absorbed in the same manner and to the same extent (subject to a minor qualification mentioned later) as the ion from other more soluble sources. Thus to take calcium fluoride as the obvious example Largent (1961)[2] has shown that in man calcium fluoride ingested as a solid was absorbed to the extent of 62% as against 96% when in solution[3]. Only the fluoride absorbed through the gut into the blood stream has physiological significance; the efficacy of a given compound therefore is proportional to the degree in which it releases the dissociated ion.

96. The absorption of fluoride ions from a dose of soluble fluoride salts is rapid and nearly complete. It may, however, be reduced slightly by the presence of other elements in the diet such as calcium, magnesium or aluminium that can complex with fluoride to give insoluble forms, thus hindering absorption and increasing faecal excretion. However, in normally composed drinking water and food this interference is so small that the view has been expressed that it can be neglected[1].

After reviewing a number of modern authorities Cremer and Buttner[1] conclude:—

> 'The effect of various inorganic ions on fluoride absorption seems to be more of an academic interest. In common human nutrition the normal mineral concentrations will be rarely in such ranges at which the fluoride absorption may be affected significantly.'[1].

97. When a liquid containing fluoride in solution is drunk a small amount is retained by the oral fluids and may be incorporated into the tooth structure by surface uptake but the greater part of the dissociated fluoride ion is rapidly absorbed by simple diffusion [4] through the walls of the stomach and the gut. The use of the radioactive isotope $^{18}F^-$ has given a great deal of certainty to quantitative data on the rates of absorption and these indicate that the maximum plasma concentration may be reached in less than one hour[5]. The total percentage of the dose absorbed in this fashion is high, being estimated at 80% of the intake from all dietary sources[6,1].

98. Once absorbed through the membranes of the stomach and gut, fluoride passes into the blood plasma and is rapidly distributed throughout the body. It is able to pass freely through capillary walls and thus to reach the extracellular fluids of all organs and tissues[7]. It can pass cell boundaries and reach the intercellular fluids but fluoride concentration in these fluids is less than that of the plasma and extracellular fluids[8, 9]. Distributed in this fashion fluoride becomes available to the skeletal structures of the body, i.e. the bones and teeth in which it may be retained and stored in proportions which, generally speaking, increase with age and with intake. The mechanisms by which this is achieved are discussed later. It may also be retained in other centres of calcification, such as the aorta, the placenta and in urinary calculi, but in these cases the retention of fluoride is secondary to the calcification and not a cause of it. (See Chapter 39—'Ectopic Calcification'.)

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Section (b)—The Blood

99. The plasma role in the distribution of fluoride is that of transport. Ingested fluoride is rapidly cleared, the excess over normal physiological levels being either taken up by the skeleton or excreted via the kidney. The plasma maintains a constant low level of fluoride and does not retain it as do the bones and teeth[1]. This level which, generally speaking, is from 0.14 to 0.19 ppm is maintained at all levels of habitual intake other than grossly abnormal[2]. Following concentrated dosages the blood value reaches a peak in about 30 minutes to one hour and thereafter falls with exceptional promptness[3]. Perhaps a maximum of 8 to 10% of an ingested dose of radiofluoride may be present for a brief time in the whole plasma volume[4].

100. The plasma fluoride is regarded as the most reliable indicator of the fluoride level of body fluids. The ability of the body to maintain it at a constant level even in the presence of elevated intake is an important factor in the fluoridation controversy and has been clearly demonstrated by epidemiological and clinical studies. The blood plasma of residents of five centres in which the fluoride content of the communal waters varied from 0.15 to 5.4 ppm was studied by Singer and Armstrong [2] in 1960. In four of the centres in which the water contained 0.15 to 2.5 ppm fluoride, the mean plasma level of the subjects tested lay within the range 0.14 to 0.19 ppm, indicating an effective bodily mechanism for maintaining a constant fluoride level in body fluids notwithstanding variations in intake. Only when the level of the water was raised to 5.4 ppm was a slight but significant raising of the serum level to 0.26 ppm noticed. Another study undertaken under conditions of abnormal intake related to patients with metabolic bone disease who were treated daily with 50-100 mg of sodium fluoride for 10 to 34 weeks. In one patient the plasma

level reached 1.8 ppm on the fourteenth treatment day but, though he continued to receive 50 mg daily it declined to 0.45 and 0.48 ppm after 33 and 34 weeks of treatment[5]. The same regulatory mechanism has been demonstrated in nephrectomized rats fed with water fluoridated at 100 ppm [6] and in the reverse direction in animals with experimentally induced hyperparathyroidism[17]. It has been estimated that an oral dose of 1 mg of fluoride (equal to the amount contained in 1 litre of water fluoridated at 1 ppm) could be expected to produce a transient rise only of 0.03 ppm in the plasma fluoride content[7, 8]. At this stage the conclusion is plain. There is no reason to believe that fluoridation of drinking water at 1 ppm will do anything significantly or materially to elevate blood serum levels above the normal range. Abnormal conditions of diet and health are considered later.

101. Dr Sutton in evidence [9] called attention to an article by Singh [10] in which whole blood fluoride levels of 0.5 to 6.1 ppm (with an average of 1.5 ppm) are claimed to have been found in subjects with skeletal fluorosis in India. The object of the reference, which was not I understood a studied one, was to indicate doubt in relation to the work of Armstrong and Singer and the conclusions of the preceding paragraph. I have read the article referred to which was a slight one and which appears to be a summary of work reported elsewhere by the same author. I have referred to some of these other reports[11, 12] and to other papers by the same author which were produced to the Commission. In one of them a whole blood concentration as high as 14.5 ppm is claimed with a general range in other subjects from 2.0 to 10.1 ppm. These, of course, are whole blood not plasma determinations and in the words of Singh himself 'Blood determinations are difficult and often inaccurate, so that measurements of blood fluoride are a poor index of absorption'[13]. This I think is generally accepted[7, 14, 15] and whole blood figures would not now be relied on in preference to values for plasma[7]. Armstrong and Singer in the last reference cited suggest that some at least of the levels claimed would not be compatible with life. I have also referred to the discussion of Singh's work in this respect by the South African Commission[16]. In particular I refer to the summary of relevant fluoride determinations derived from Singh and which appears as Table IV-II of the South African Report. The inconsistencies and inherent improbabilities of the figures cited are considerable and their only effect is to reinforce the doubts expressed by Armstrong and Singer as to the suitability of whole blood as a fluoride indicator and the reliability of the tests and methods employed. Neither of these two documents on which I have relied would of course have been available to Dr Sutton but in any event I am satisfied the reference was ill considered. In any case, whatever the value of these reports is, they have no relevance to fluoridation at 1 ppm.

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Section (c)—Soft Tissues

102. Fluoride is not, as it is in bones and teeth, stored in the soft tissues of the body except that elevated levels may be found associated with centres of calcification. Transient concentrations may also be found in the gut and in the kidney in the course of absorption and excretion.

103. In the data relating to this subject analytical difficulties are apparent not only in the early work on the subject which should, generally speaking, be treated with caution[1, 2] but in the essential techniques employed[3, 4]. It is obviously difficult to compare with any accuracy analyses of the fluoride content of fresh organs (where the concentration will reflect the fluid content of the organ including the variable content of the whole blood fraction) and those of similar organs dried and ashed before analysis[3, 4]. In the latter the concentration will be higher than and will not match the concentration in the live or the fresh organ. Ectopic calcification may also be found in some glands and tissues not normally associated with calcified deposits, such as the thyroid[5, 6, 7] and these adventitious deposits can inflate values found on analysis.

104. These factors have made for uncertainty reflected in the attention paid to this relatively unimportant topic by both proponents and opponents of fluoridation, the one alleging that the soft tissues do not store fluoride, the other alleging that they do and pointing to reports of fluoride content which, particularly in the case of dried organs, are greater than might be expected, did such tissues merely mirror plasma levels.

105. There are really two questions involved, the first being what are the normal concentrations of fluoride in soft tissues and the second, whether fluoridation is likely to increase them. I take no account here of the kidney which is dealt with separately under another chapter and which has the ability temporarily to concentrate fluoride in its excretory role.

106. As to the first question the difficulties already referred to make the interpretation of the available data problematical. This can be quickly seen if one compares the values for normal fresh tissue given by Matuura *et al* (1955)[8], Gettler and Ellerbrook (1939)[9] and Herman *et al* (1958)[10].

I reproduce the following comparative table from Smith[11].

TABLE IV

NORMAL CONCENTRATIONS OF FLUORIDE IN THE ORGANS OF MAN

(*PPM F IN FRESH TISSUE*)

Organ or tissue	Matuura *et al.* (1955)	Gettler and Ellerbrook (1939)	Herman *et al.* (1958) *
Bladder		0.40—0.68	15—185
Brain	1.8—4.3		
Fat	3—4		<5—145
Gall bladder	3.9		
Hair	14—30		13—171
Heart	1.3—6.6	0.44—0.60	
Intestine	2—8		
Kidney	2.1—94	0.56—0.78	<5—181
Liver	2.5—19	0.36—0.70	
Lung	4.5—23	0.16—0.42	
Marrow	44		
Muscle	2—3.7		
Nails			10—186
Nerve	16		
Pancreas	2.8—38		
Prostate gland	—		<5—86
Skin	5		10—290
Spleen	2.6—18	0.25—0.30	
Stomach	2.9—6.7		
Tendon	24		

Taken from Smith[11]. * Concentration basis not stated; assumed by Smith to be fresh weight.

Matuura incidentally gave a value of 10 to 18 ppm as the normal fluoride concentration in blood as against 0.40 to 0.68 ppm found by Gettler and Ellerbrook. It is suggested such values would be lethal[2]. Obviously the differences between these figures reflect something more than standard variation but the real point is as Smith points out[12], the quest for a normal figure; when the difference between normal and abnormal may be only a few ppm or even fractional values of such a part; when dealing with matters of such analytical difficulty; when allowance has to be made for normal variation as between individuals (age, sex, previous exposure, all being taken into

account) and as between different organs both in the same and in different individuals (apparently frequently found); all these matters added to the technical difficulties of the subject make this quest for a normal figure rather illusory.

107. There is no doubt that fluoride is a normal constituent of all body tissues. The generally accepted opinion would seem to be that in the absence of adventitious factors, such as calcification, the concentration found in the soft tissues and organs of the body does no more than reflect the concentration in the blood. Its content will of course be determined by the volume of the blood and other body fluids that may have been present in the living fresh state. Smith, whose treatment of the matter is detailed and the most satisfactory, concludes in relation to such tissues and organs:—

> 'It is apparent that no organ has a particular and lasting affinity for fluoride; in general, tissue concentrations reflect the blood levels.'[13]

Further, while whole blood levels, reflecting perhaps the variable content of the hematocrit, are generally higher than those of the plasma or extracellular fluids, there is evidence of a relatively restricted entry of fluoride into the intracellular fluids[14]. It is accordingly thought that the total tissue fluid content of fluoride would be lower than the plasma or blood levels in all organs and tissues except those such as tendon or cartilage where calcification may be present[15].

108. Apart from calcification and excretion, I have found no satisfactory evidence of storage or retention in any soft tissue. Such levels as have been reported in excess of blood or serum levels (generally in respect of dried specimens) are slight (a few ppm as contrasted with the hundreds or even thousands found in calcified structures) and modern studies with the radioactive isotope F^{18} both in animals and humans to which both Smith[13] and Armstrong and Singer[2] refer at length have failed to indicate any significant retention. The experiments of Appelgren *et al* (1961)[16] are constantly referred to[6]. In these, using radioactive isotopes of both calcium and fluoride, he traced the uptake of both elements, the fluoride exhibiting an affinity for the hard tissues while the calcium was found in significant quantities in some soft tissues.

109. As to the second aspect of the matter, a study by Smith *et al*[17] found no significant differences between desiccated human tissues taken from subjects drinking water fluoridated at levels ranging from 0.0 to 4 ppm. Armstrong and Singer[2] and Smith[3] all consider that if this data be extrapolated so as to make an approximate comparison with fresh tissue values, then there is substantial agreement between the values so obtained and those of Gettler and Ellerbrook (*supra*) and they would indicate a fluoride content of 0.5 to 1.0 ppm in normal fresh human tissue. Call *et al*[18] studied the dried tissues for two groups of subjects with high and low exposure from atmospheric sources in industrial areas. There were no significant differences between the mean or average values obtained for either group, though the range in the high fluoride group tended to be wider in that the highest figures obtained for individuals studied were, generally speaking, to be found in the high fluoride group. But there was no consistent trend either as between individuals or as between organs in the same individuals. No age corrections were attempted, although there was a considerable difference between the mean ages of the groups; the fluoride group being considerably older than the other. Call's study is cited by the opponents of fluoridation but his own conclusion was that the results demonstrated that fluoride is not stored in significantly increased amounts in soft tissues when the degree of exposure is increased.

110. However, if the exposure to fluoride of the subject is sufficient to raise the serum level[19] it is to be expected that this would be reflected in an increased level in organs and tissues but plainly there is no arithmetical equivalence and the effect is slight at anything but lethal levels. This has been demonstrated in a great number of animal studies listed by Smith[20]. These studies and an even more dramatic comparison made by Gettler and Ellerbrook between normal dogs and dogs given massive doses of fluoride for two weeks (equivalent to 1-2 grams of fluoride in a man) show that the increase is not proportional to intake and they demonstrate clearly the remarkable ability of the body to regulate blood and tissue fluoride at constant levels.

111. Difficulties in the data have been referred to. Taking them into account there would seem to be no reason to doubt that the soft tissues of the body do not retain or store ingested fluoride in significant quantities.

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Section (d)—The Skeleton

112. While the body tends to maintain a constant fluoride level in soft tissue and blood, apart from transient concentrations in the gut and kidney in the course of absorption and excretion, the bones, teeth and other areas of calcification within the body attract and retain fluoride. To that extent it is correctly said that fluoride is a bone seeker.

113. Different levels of absorption and concentration have been demonstrated for different types of bone, e.g. as between rib and vertebra, cancellous and compact as they have for different parts of the tooth, i.e. as between cementum, dentine and enamel. It is not proposed here to discuss those differences, the purpose being to inform generally only of the mechanisms whereby it is thought the fluoride ion is taken up and retained by the skeletal tissues of the body.

114. About 96% of the fluoride found in the body is deposited in the bones[1]. The accumulation is maximal during bone formation and growth. After growth ceases the fluoride content of bone continues to rise but more slowly than before. In this regard the fluoride ion is said to be unique in that it continues to deposit in the calcified structures after the other constituents of bone have reached a steady state[1]. In this it may be said to be a cumulative substance.

115. In subjects with no prior exposure to fluoridation, the immediate rate of accumulation will be high, but a steady state is soon reached and once reached experimental data suggest that at levels of intake up to 20 ppm, about half will be retained within the skeleton [2] and the remainder excreted, but the proportion retained decreases with age[3].

116. However not all fluoride accumulated by the bones is retained indefinitely. Concurrently with uptake and growth, there is a continuous process of release and resorption due to physiological processes involved in the remodelling and replacement of bone. In such processes, fluoride previously incorporated in bone is released and may be either excreted or possibly reabsorbed at other bony sites nearby.

117. Subjects who have previously had a high fluoride intake will, on changing to a lower level, continue to exhibit for a prolonged period a urinary level of fluoride higher than their water intake which, in a state of balance, it normally matches. Thus bone not only stores fluoride but may be considered and is so described as a reservoir of fluoride and should the bodily supply be diminished, fluoride will be mobilised from the skeleton and pass into the body fluids[4, 5, 6, 7, 8].

118. In relation to non-toxic levels of ingestion, several writers have demonstrated a linear relationship between the amount of fluoride deposited in bones and the amount ingested. Largent (1961)[2] has claimed that so long as the level of ingestion remains unchanged, any further storage will eventually be offset by the mobilization of previously stored fluoride: thus a virtual balance will be reached between absorption and storage on the one hand and mobilization and excretion on the other[9]. Largent's subjects included some whose water supply was as high as 20 ppm. Others maintain the view that in about the sixth decade a plateau is reached and thereafter storage decreases as resorption and excretion tilt the scale[10, 11].

119. Normal physiological changes associated with age must be taken into account but their effect is difficult to assess. Cessation of growth will decrease deposition but remodelling and resorption may increase deposition in adjoining structures by mobilization and secondary deposition[1]. However, more recent work reviewed by Weidmann and Weatherell[9] tends to leave the question of a plateau an open one. So long as ingestion rates are unchanged, the balance of opinion seems to be in favour of a gradually decreasing rate of accumulation, certainly lessening with age but not ceasing. The fullest review of the subject is perhaps that contained in the South African Report which concludes—

> '. . . for any constant steady intake of fluoride a predictable equilibrium concentration of fluoride exists in bone. The balance of this equilibrium is not complete inasmuch as, with advancing age, bone fluoride concentration increases slowly and any factor which gives rise to new bone formation will displace the equilibrium in the direction of further skeletal retention'[12].

120. If the ingestion rate is changed, e.g., by the introduction of fluoridation in a community which previously drank unfluoridated water, then an increased uptake is to be expected even in the aged. This is indicated by the studies of Zipkin[13] in particular in which he studied bone fluoride concentrations in subjects whose exposure to water containing 1 ppm of fluoride began at the average age of 63 years. In those relatively old bones, with a length of exposure not exceeding 12 years he found a mean fluoride concentration of 1460 ppm as against an average value of 490 ppm in individuals of an average age of 56 years who had been drinking water with an average fluoride content of 0.2 ppm.

121. The persistent accumulation of fluoride in bone is a natural phenomenon. It has been demonstrated in virtually fluoride-free areas, though certainly the higher the intake the more fluoride will be accumulated. I borrow from the South African Report[14] the following tables prepared from data collected by Zipkin *et al*[13] and Zipkin[15] as a simple illustration of the relationship between age, fluoride intake and skeletal levels.

TABLE V-I

SKELETAL FLUORIDE CONTENT IN RELATION TO AGE AND FLUORIDE INTAKE

Fluoride Concentration in Drinking Water (ppm)	<1.0	1.0	2.6	4.0
Average Age (yrs)	55.5	75.6	65.9	56.0
Average Period of Residence (yrs)	28.1	21.5	32.9	21.3
Iliac Crest Fluoride in ppm (ash)	720	2,250	4,500	6,870
No. of Cases	20	4	25	4

From Zipkin[15].

TABLE V-II

SKELETAL FLUORIDE CONTENT IN RELATION TO FLUORIDE CONTENT OF THE DRINKING WATER AND DURATION OF EXPOSURE

Fluoride Concentration in Drinking Water (ppm)	<1.0	1.0	2.6	4.0
Duration of Exposure (yrs)	10-87	10-50	10-76	10-48
Fluoride in Dry Fat-free Bone (ppm mean ± SE)	483 ± 50	1460 ± 200	2700 ± 230	4073 ± 680
No. of Cases	33	5	27	4

From Zipkin *et al*[13]. Taken from Report of South African Commission[14].

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Section (e)—The Teeth

122. The incorporation of fluoride in the teeth is basically similar to that in bone. It is most rapid during the time of formation and mineralisation which, to cover both the deciduous and permanent dentition may extend from the fourth month of uterine life to the 16th year. If the wisdom teeth be ignored (they are sometimes congenitally absent or eruption is delayed) then the end of the 8th year probably marks the limit of this period so far as the enamel is concerned. It will be supplemented by a further period of high intake during maturation. This process is most active in the pre-eruptive stages, but is significant for a short time thereafter. Taking all these factors into account from birth through to the end of the 15th year of life (which will also cover the period of root formation) may be taken as the total period during which the uptake of fluoride by dental structures is at its maximum.

123. Erupted teeth differ from other skeletal tissue in that once formed, with the exception of the dentine, there is no remodelling, and in the case of the external covering or enamel no cellular activity, and subsequent uptake of fluoride is modified by these factors. Hence the importance of the pre-eruptive phase of the dentition in relation to fluoride uptake. It should not be assumed, however, as is sometimes done, that all possibility of uptake from fluoridated water ceases after the teeth appear in the mouth.

124. As with bone, the fluoride concentrated in the teeth can be shown to bear a constant relationship with the level of fluoride in the drinking water and the age of the subject[1, 2]. Not being subject to remodelling teeth do not tend to lose fluoride by resorption, except in the case of root structures in deciduous teeth particularly[3] but they may lose fluoride due to the loss of tooth substance from attrition[4] or possibly by reversal of the process of ionic exchange due to a fall in the fluoride concentration of the oral fluids[5, 6, 7].

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Section (f)—Methods of Uptake by Hard Tissue

125. Fluoride incorporated in hard tissue during the period of formation and crystal manufacture is said to be taken up by accretion. Fluoride so absorbed is taken up by cellular activity and may be found in deeper or denser layers of tissue to which, after formation, the penetration of body fluids may be difficult. This may be taken to represent the initial phase of fluoride uptake in hard tissue, but the general consensus of opinion is that the main mechanism whereby fluoride is taken into these tissues, is by a process of ionic exchange, in which the dissociated fluoride ion exchanges with and takes the place of other ions in the crystal lattice of the structure concerned. This is referred to as incorporation by exchange.

126. This will also be maximal during the period of formation and growth as the hard tissue crystallites are at this stage smaller in size, greater in number and offer a greater surface area to the extracellular fluids of the body. In addition these younger tissues are more permeable and more vascular.

127. But unlike the process of accretion, ionic exchange may continue after the initial phase of formation and mineralisation, because it is dependent, not upon the biological activity of bone or enamel forming cells, but upon a chemical reaction at the surface of the bone or enamel crystal, which is independent of cellular activity. This may be seen in laboratory experiments whereby if bone or enamel crystals are ground up and mixed with fluoridated water in a test tube, analysis will disclose an uptake of fluoride into the calcified tissue. This process is, however, dependent on access by the fluoride ion to the site of exchange; this is by the body fluids, the blood or the saliva.

128. After formation but before eruption, the maturing teeth, contained in the tissue of the gum, are freely accessible to the circulating body fluids and uptake by ion exchange is high as a result. The importance of the pre-eruptive stage as a whole can be gathered from the fact that Brudevold[1] has calculated that the presence of 1 ppm of fluoride in the water supply causes an increment of about 300 ppm in the surface enamel over that occurring in teeth from low fluoride areas.

129. Bone may remain biologically active throughout life, though activity decreases with age. Hence in the case of bone the process of accretion remains significant. Dental enamel differs, however, in that once formed there is no remodelling and no cellular activity. This may persist however in the formation of cementum (the external bony layer of the tooth below the gum) and of secondary dentine.

130. As the process of uptake by accretion is not significant in enamel after formation any post-eruptive uptake by the surface layers of this part of the tooth can only be from the oral fluids or saliva. These may penetrate to the dentine below the enamel either through naturally occurring fissures or through cavities due to disease or trauma. The dentine, unlike the enamel, may also achieve a small uptake through the circulating fluids of the body conveyed to it by the vessels of the pulp.

131. Another method of distinguishing between the two methods of uptake (frequently used in the evidence) is to refer to them as systemic, denoting incorporation in the course of development and topical to denote surface ionic exchange whether pre or post-eruptive. This has led to some confusion, because ' topical ' is also popularly used to describe the method of direct oral application such as the surface effect achieved by drinking fluoridated water or by painting the teeth with fluoridated solutions or by the use of a dentifrice or a mouth wash.

132. The dependence of teeth on ionic exchange for any uptake of fluoride subsequent to formation and calcification, means that as age increases, the surface layers in contact with fluoride-containing fluids, will achieve higher concentrations than those in the interior of the structure. This is true particularly of the outer layer of the enamel in contact with the oral fluids and the innermost layer of the dentine where at the dentino-pulpal junction it is in contact with the vascular tissue of the pulp. Brudvold *et al.* (1956)[2] has shown that the gradient can be a steep one and this is a factor of some importance in considering the published results of some experiments where no attempt has been made to relate the sample analysed to its relative distance from the fluoride-rich surface.

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Section (g)—Chemical Mechanisms of Fluoride Uptake

133. Two mechanisms have been described in general terms whereby the fluoride ion is incorporated in teeth and bone. They represent the commonly held views on the subject. However, while incorporation in fact cannot be doubted, there are still uncertainties as to the exact chemical paths by which this is effected, deriving no doubt from the fact that the ultimate secrets of bone formation and composition are still matters of speculation.

134. The mineral constituent of the hard tissues (teeth and bones) of the human body is presumed to be an apatite with the generic formula of $Ca_{10}(PO_4)_6(OH)_2$, Kay *et al.* (1964)[1]. It is called generally hydroxyapatite. But besides the hydroxyl ion, other ions—notably carbonates, magnesium, manganese, and citrate—are known to occur on the crystal surface and may be incorporated in the crystal lattice, giving rise to a variety of biological apatite crystals which can only be distinguished with difficulty. Because of its physical characteristics, the fluoride ion by hetero-ionic exchange can readily take the place within the crystal lattice of the hydroxyl group and this, by a three-stage process described by Neuman *et al.* (1958)[2] would seem to be the principal route whereby fluoride is incorporated into the hard tissues of the body as fluorapatite or more strictly as fluorhydroxyapatite as it is usually a mixed crystal.

135. It can substitute also for bicarbonate ions[3] and possibly citrate and some others[4, 5] but not, it should be noted, for calcium, phosphate and chloride[6, 7, 2, 5]. If the statement so frequently repeated that fluoride is a 'calcium robber' is meant to convey that it is capable of substituting for the calcium ion in bone salt and thus displacing it, the answer is that according to known physico-chemical laws this is not possible[8, 9, 2]. In fact the literature reviewed by Hodge and Smith[10] would indicate either no change or an increase in calcium percentage in bone with increasing fluoride intake.

136. The evidence is that the fluorapatite crystal so formed is stronger and more perfectly formed than the hydroxyapatite crystal which it may replace. This was stressed in the evidence of Professors Storey and Martin. It is supported by the work of Posner (1963)[11] and Taves and Neuman (1963)[12] and by the evidence both clinical and theoretical that fluoride exhibits a beneficial influence on the morphology of the teeth; they are denser and better formed and more resistant to attack by acid, including the acid products of carious processes. These effects are generally accepted[13]. They will be referred to in more detail when the subject of caries is being discussed.

137. Lastly as the role of enzymes in physiological processes and the influence of fluoride upon them has been mentioned it is proper to note the categorical statement by Hodge and Smith (1965)[14] in relation to the deposition of fluoride in bone generally:—

> 'Essentially none of the fluoride is attached to the organic part of the bone. Von Fellenberg and Schmid (1949b) and Peckham *et al.* (1955) drew similar conclusions for the organic part of dentine.'.

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Section (h)—Excretion

138. Insoluble fluorides on ingestion will not be absorbed and will be excreted via the bowel. A small and relatively unimportant percentage of soluble fluorides may also be lost in the same way due to either complexing in the gut or by reabsorption by the bowel from the body fluids.

139. Ingested soluble fluorides will pass almost wholly into the body fluids through the walls of the stomach or the small intestine. The body, as has been seen, tends to maintain a constant level in the blood and body fluids and this is achieved in relation to any excess by bone deposition and urinary excretion. In hot weather the amount excreted through perspiration can also be significant.

140. Urinary excretion is rapid and efficient. With single doses of 1.5 to 5.0 mg of fluoride 20% or more will be found to have been excreted within the first three hours, the highest rate being within the first hour[1, 2]. The excretory mechanism that is principally involved is that of direct glomerular filtration and not that of tubular reabsorption. This enables the kidney to handle fluoride almost as easily as water[1] and at least 100 times more effectively than chloride[3, 4, 5, 6] and in all but advanced forms of kidney disease[7, 5].

141. The amount excreted will depend to a large extent on the age of the subject, the degree and duration of previous exposure and the state of skeletal saturation. In an individual not previously exposed to fluoride a larger percentage of a given dose (up to 50%) will be deposited in bone and less excreted[8, 9, 10]. The amount deposited will lessen and the amount excreted will increase with age[11, 12] as exposure at a constant level continues[12]. Ultimately an approximate steady state of skeletal balance will be reached and though deposition will continue it will be on a much reduced scale, and will to some extent be set off by fluoride released into the body fluids by osteoclastic reworking of the skeleton. So long as the steady state is maintained the urinary excretion will approximate the intake[9].

142. In such circumstances the urinary concentration has been found to match very closely the fluoride level of the drinking water[13, 14, 15, 12] which makes urinalysis a reliable indicator of the level of fluoridation, at least in circumstances where there has been no present change in residence or habit and particularly if 24-hour specimens are obtained. This obtains whether the water is naturally or artificially fluoridated[16, 10], and whether the ingested fluoride is taken as a single dose, e.g., by tablet, or in multiple doses spread over a 24-hour period, e.g., in the drinking water[17].

143. An interesting and important question is whether the rate of excretion will remain constant at different levels of fluid intake so that a higher nett percentage will be retained if the fluid intake be significantly increased or whether the homeostatic mechanisms already described are effective in such circumstances to stimulate excretion and thus maintain fluoride balance. The question is important in considering the effect of excessive thirst and consequent water consumption due to temperature or disease. The South African Commission considered the matter and state a firm conclusion that 'if the body is confronted with an increased rate of fluoride intake a compensatory increase in the rate of excretion occurs so that the extent of any retention of fluoride is controlled in such a manner as to minimise deviation from the pre-existing state.'[18]. The principal evidence in support is that of Carlson, Armstrong and Singer (1960) (a) and (b)[19, 20] and this is regarded as a safety mechanism against overdosage due to excessive fluid intake[21]. However it is clear that any such effect is not absolute and that high fluid intake due to thirst or temperature will increase nett retention, particularly if constant. The matter is considered further under succeeding chapters relating to the 'Abnormal Individual' 'Endemic Fluorosis in Native Populations' and 'Optimum Levels Determined According to Temperature'.

144. It has been clearly shown that the process of building up to a state of skeletal balance is reversible. Thus if subjects move from a high fluoride area to a low fluoride area their urine will for long periods (up to two years has been demonstrated) show higher levels than current intakes, reflecting a skeletal loss. This will continue until equilibrium between intake and skeletal deposition is again achieved[8, 14].

145. Fluoride can be excreted in the sweat. Under conditions of excessive sweating up to 50% of the total daily excretion can be excreted in this fashion[8, 22, 23]. It is considered by Hodge *et al.*[5] that to a limited extent sweat may have a role in the regulatory mechanism described in paragraph 143.

PART E—BASIC DENTAL ANATOMY AND PATHOLOGY

CHAPTER 10. ELEMENTARY DENTAL ANATOMY

Section (a)—The Dentition

146. This report is not written for dentists and will suffer in point of clarity unless some attempt is made to describe the elementary features of the human dentition. It does not attempt to match in detail the excellent summary of the same subject contained in Appendix ' A ' to the South African Report to which reference can be made if desired.

147. The human mouth is normally equipped with twenty deciduous (milk, primary or first) teeth and thirty-two permanent (second) teeth. Their formation, calcification and eruption takes place at varying times of infantile and adolescent life as can be seen from Table VI.

148. If attention be focussed on enamel formation it will be noticed that in respect of the permanent dentition practically the whole process of enamel formation takes place after birth. There is a tendency to ignore the third molars or wisdom teeth, which are at times congenitally absent, and though this may be questioned, if it is accepted it will be seen that the most important period of enamel formation is from birth till the end of the eighth year. However, after completion and before eruption, there is a period of maturation in which the tooth surface is relatively permeable and is freely accessible to the body fluids permitting uptake of fluoride by ion exchange and this if allowed for could extend the period of maximum fluoride uptake to the fifteenth year.

149. The first permanent molars are usually the first permanent teeth to erupt. They are frequently referred to as the six-year molars indicating the age at which eruption normally takes place. They have assumed a particular significance in the fluoridation literature and their loss or if retained their condition is often used as an index of dental health.

150. The composition of a tooth is as shown in Fig. I. The crown is that portion of the tooth which protrudes above the gum, the root that portion which lies below. Enamel covers the crown and cementum the root. Both enclose the dentine. Dentine represents the bulk of the tooth substance. It itself surrounds the thin central pulp chamber and root canals which contain the pulp or vascular tissue in the centre of the tooth.

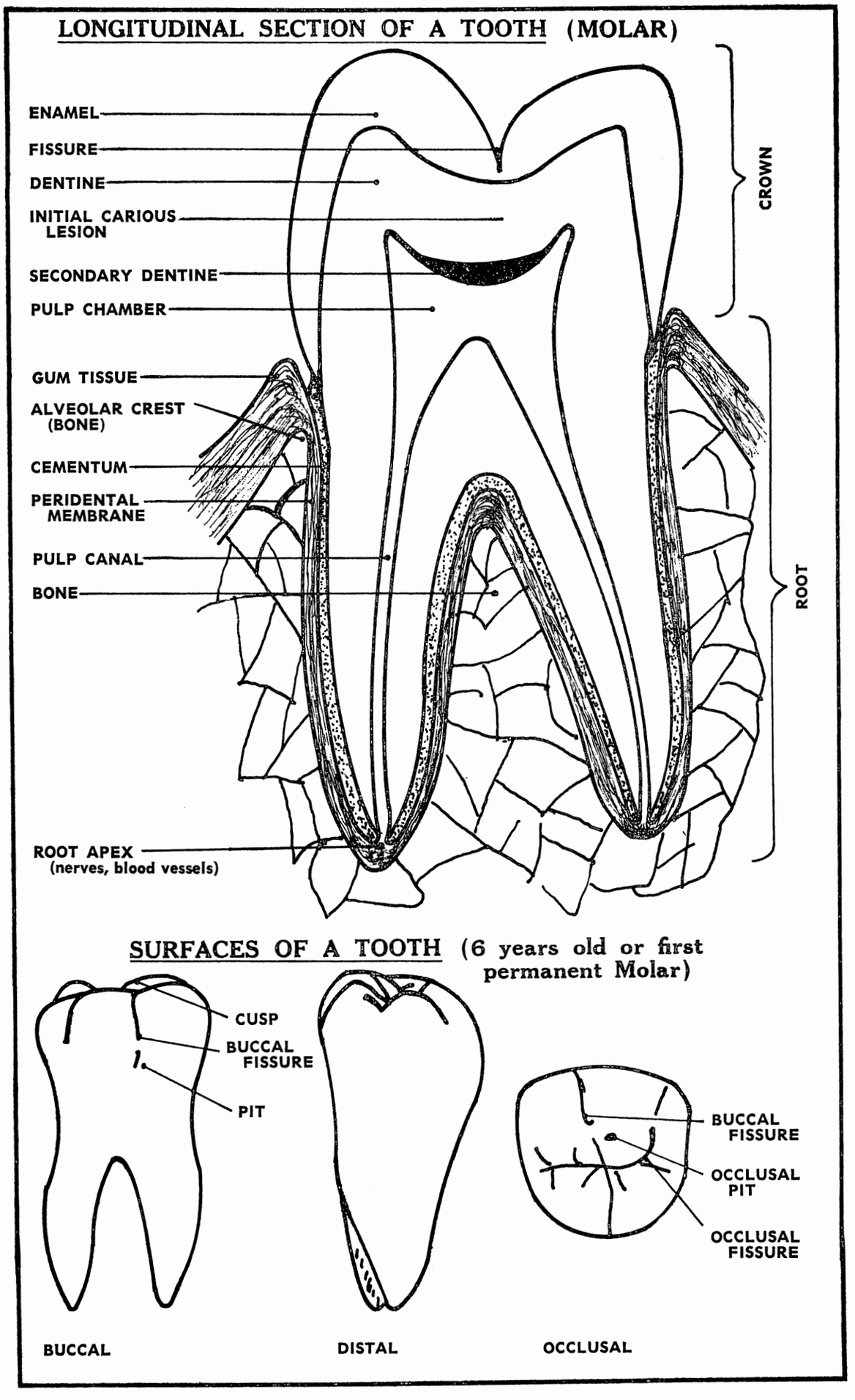
151. The teeth are attached to the alveolar bone of the jaw, not rigidly but by means of a fibrous ligament called the periodontal membrane. This means that they retain a degree of mobility and may change their position post eruptively in the dental arch or jaw. Premature loss of some teeth may allow other teeth to drift, i.e., to change position in the dental arch giving rise to orthodontic abnormalities.

152. The primary substance of enamel, dentine and cementum is apatite as in bone. The composition of bone is twofold. It has an inorganic or mineral substance contained in an organic or cellular matrix. While the mineral or apatite is common to bone, enamel, dentine and cementum, there are differences between them in the arrangement of the crystals, their denseness and in the content of organic matter. Both cementum and dentine closely resemble bone in their incorporation of organic matter, whereas enamel has very little, less than 1% as against approximately 20% for dentine.

153. Dentine differs from bone in the absence of included cells. Dentine-forming cells are however found on the pulpal surface of the dentine and these, as a protective reaction to stimulus or as a senile change, may in life form dentine within the pulp chamber thus reducing its size. This is called secondary dentine. The degree in which fluoride is taken up by the dental tissues is, in descending order, cementum, dentine and enamel but the significance of this high uptake by the cementum is little understood.

154. Distinctive titles are given to the different surfaces of a tooth. Labial (or buccal) denotes the surface towards the lips (or cheeks)—the front surface. Its counterpart is the lingual or rearmost surface. The surfaces between the teeth are the interproximal, while the biting surfaces of molars and bicuspids are known as the occlusal surfaces. On the incisors the biting surfaces are known as incisal. Interproximal surfaces are further distinguished as mesial (closest to the midline of the arch) and distal (the side furthest away from the midline). Developmental faults in enamel, found particularly in the occlusal surfaces of the molars are known as pits and fissures indicating either a sharp pointed depression or a groove. They are of some importance as supplying an early focus for carious attack.

FIG. I.


LONGITUDINAL SECTION OF A TOOTH (MOLAR)
ENAMEL
FISSURE
DENTINE
INITIAL CARIOUS LESION
SECONDARY DENTINE
PULP CHAMBER
GUM TISSUE
ALVEOLAR CREST (BONE)
CEMENTUM
PERIDENTAL MEMBRANE
PULP CANAL
BONE
ROOT APEX (nerves, blood vessels)
CROWN
ROOT
SURFACES OF A TOOTH (6 years old or first permanent Molar)
CUSP
BUCCAL FISSURE
PIT
BUCCAL FISSURE
OCCLUSAL PIT
OCCLUSAL FISSURE
BUCCAL
DISTAL
OCCLUSAL

tective or essential dietary element has been removed in the process of refining natural foods to suit the modern palate. Precisely what is not accurately known, e.g. it is not known why the free use of sugar-cane does not promote caries while refined white sugar does, though phosphates have been implicated[16]. But the fallacy lies in the isolation of a single factor to the exclusion of others in a fultifactorial problem.

162. For if diet be considered, not only as a negative factor resulting in nutritional deficiency but as a positive element influencing the oral environment then there is general agreement as to its importance in the production of caries. From this point of view there is hardly any dissent that the refined sticky carbohydrates so prominent in the modern diet and so beloved of childish appetites are grossly implicated in the initiation of carious attacks. A more savoursome description is ' a combination of a sparingly soluble glutinous polysaccharide with a more diffusible soluble sugar '[17]. The alliteration I am sure is coincidental.

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Section (b)—Carious Lesions, How Produced

163. The essential lesion of caries is demineralisation of the inorganic part of the teeth with destruction of the organic leading to total loss of substance. There are two major theories as to the means by which this is achieved.

164. The first is the chemico-parasitic theory of Miller, the second the chelation proteolysis theory of Schatz. The first postulates the production of acid by microbial fermentation of carbohydrate residues in the mouth. The acid so produced decalcifies and dissolves the inorganic part of the tooth. Destruction of the organic residue follows as a secondary consequence as a result of proteolytic enzymes derived from bacteria. The second theory would reverse the order. Bacterial action upon the organic component of teeth is supposed to produce chelating or combining agents which have the property of leaching out the calcium ion from the inorganic apatite thus leading to decalcification and destruction.

165. Both theories presuppose bacterial action as an essential part of the caries producing mechanism and this, in the light of the experiments of Fitzgerald and Keyes (1960)[1] and Orland, Blayney and Harrison (1964),[2, 3] is accepted. In the opinion of Professor Thonard it is highly likely that both mechanisms, and possibly others, play a part in the production of caries but professional opinion and the great bulk of academic opinion would certainly favour the acidogenic theory of Miller as the most important. Of this matter the South African Report concluded that there was 'considerable evidence in support of the acidogenic theory' while the second was 'subject to extensive criticism'. At least one writer of authority (Brudevold) speaks of the evidence in favour of the acidogenic theory as overwhelming[4].

166. As to the type of bacteria responsible Professor Thonard in his submission reviewed the evidence to indicate that two groups of micro-organisms are almost certainly implicated, viz., the streptococcus and the lactobacillus[5]. They share the ability to convert glucose to lactic acid but differ in other characteristics notably, in this context, in the ability to live and multiply at different levels of acidity. Arising from this and other matters, the possibility is suggested that the cariogenic properties of these two groups may be cumulative and not alternative, i.e. that the lactobacillus may take over when the pH of the carious lesion has reached a level which inhibits the streptococcus. The possibility that other groups and other organic acids may be implicated cannot be excluded.

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Section (c)—Multiple Roles of Fluoride in Caries Resistance

167. Dental plaque has already been mentioned. It is rich in the micro-organisms implicated and accumulates most readily at the same sites as do the carbohydrate and other food residues retained in the mouth. Bacterial fermentation of these residues produces lactic acid and perhaps other organic acids as well by complicated chemical paths, but of which one essential step is the production of an enzyme enolase.

168. In this respect research of the last ten years has indicated that the fluoride ion may act by inhibiting acid production as well as by increasing the resistance of teeth to acid dissolution by influencing tooth composition and structure. This it does by inhibiting the enzyme referred to. However this role is dependent upon fluoride being present in concentrations not known to be achieved in the body fluids, viz.: from 8 to 10 ppm. This level is not possible in the saliva except directly by the ingestion of a fluid of that or greater strength. However, the work referred to[1] suggested a hitherto unsuspected and specific role of the dental plaque in that it has been shown to be capable of concentrating the fluoride ion in proportions which greatly exceed those of normal serum or salivary concentrations. Such concentrations are capable of inhibiting the formation of enolase in the fermenting carbohydrate substrate on the tooth surface, thus breaking and inhibiting the further progress of the glycolytic cycle which leads to the production of lactic acid.

169. The release of the plaque stored fluoride would seem to be a function of an acid pH, thus it would seem probable that the plaque fluoride exerts its maximum effect in the presence of acid, i.e., when it is most needed[2]. It only remains to add that it is believed that the plaque fluoride is derived from the saliva and the drinking water rather than from the enamel surface[3] which it overlies and that studies comparing plaque fluoride concentrations of subjects from fluoridated and non fluoridated areas have shown higher and more effective values in the former.

170. Lastly, it is now suggested that fluoride not only provides a tooth mineral in itself more resistant to demineralisation by acid but stimulates a reparative or restorative process of remineralisation in the carious lesion when it occurs. The production of acid at the tooth surface is not constant but intermittent and 'an alternating process of demineralisation and reprecipitation takes place'.[4, 5, 6] The ability of fluoride to induce apatite formation from solutions of calcium and phosphate and thus by remineralisation and reprecipitation of apatite to assist in maintaining

the status quo of the mineral structure of the tooth is regarded as yet another crucial role for fluoride in caries resistance[7, 8, 9]. It is of particular importance in retarding the incipient lesion of early caries[10].

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Section (d)—Other Methods of Caries Control

171. The recognition of the part played by bacteria in the production of caries naturally raises the question whether control of the carious process can be achieved by eradication of the micro-organisms responsible. Professor Thonard has reviewed the evidence and I accept his conclusion that, while such measures have a theoretical interest, they offer no present practical solution; certainly none superior to the fluoridation of drinking water.

172. In his view there is no evidence to support the hypothetical possibility of an acquired immunity developing or being developed as a protective mechanism, indeed the evidence suggests the contrary, as a drop in immunity has been demonstrated in successive generations of germ-free rats fed with high carbohydrate diets and the same strain of human lactobacillus[1].

173. Control by mechanical means or by chemotherapy has at the moment but limited possibilities. Theoretically any measure that will reduce plaque, food debris and bacterial concentrations can do nothing but good. It is plain that brushing as commonly practised is largely ineffective owing to the inability of the brush thoroughly to cleanse the most susceptible areas and because of the common failure to use the tooth brush immediately after meals[2]. However differences in caries prevalence have been demonstrated between children with good and neglected dental cleanliness[3] so this is not to suggest as a practice it should be abandoned. Chemotherapy has been employed by the incorporation of straightout bacteriophages in dentifrice preparations but without significant effect[4]. Slightly better results appear to have been achieved by the incorporation in a dentifrice of substances such at laurolyl sarcosinate which has both an enzyme inhibiting and a detergent effect[5]. Chemotherapy is however subject to severe limitations. It must be in a form capable of being tolerated by mucous membrane; it must persist beyond the period of application (it is said that oral fermentation may be observed within five minutes of the ingestion of carbohydrates) and if it is to be in antibiotic form then it must guard against the danger of species immunity, i.e., that a resistant flora may be evolved and establish itself within the mouth, which could have serious results, e.g., by initiating an attack of thrush.

174. Whatever possibilities may exist and Professor Thonard has I think in this field canvassed most of them, his evidence and the other evidence given before me which I accept supports strongly the conclusion that such measures are often impractical and so far as they are practical they do not supplant the importance of strict dietary control and fluoridation which remain the only proven means of lowering both the prevalence and severity of caries.

175. For it is important to make this point. No witness before me has sought to minimise the importance of dietary restraint as a means of controlling caries. An experiment in a N.S.W. boys' home[6] has shown that under conditions of institutional discipline a rigidly controlled diet is capable of promoting a high degree of freedom from caries. Other institutional successes are listed by Snyder[7]. The effect of the 1939-45 war on the dietary habits of some European and other countries produced similar results. United Kingdom studies showed a steady decrease in

the prevalence of caries which continued until 1947-48, by which time the prevalence began to rise[8, 9]. Comparable results have been reported for Scandinavia, Finland and Japan. Reviewing the literature, Hartles concludes, 'The factor which was common in all cases was a reduced consumption of sugar and manufactured sugary confections[10, 9].

176. But it is the despair ever of enforcing a national dietary discipline adequate to eliminate the cariogenic factors in our established civilised diet that produces so much of the fervour with which the solution of fluoridation is sought, particularly by members of the dental profession. No one has sought to say that if fluoridation is introduced diet may be disregarded. The two are properly regarded as supplementing one another, not as alternatives. One acts to increase resistance and the other to weaken the attacker, but it is established that negative resistance simply due to the absence of deleterious material in the diet will not survive the first accumulation of fermenting carbohydrate unless the diet has contributed a positive element augmenting resistance as well. This was illustrated very vividly by the studies of the Tristan da Cunha islanders[11].

> 'Here the introduction into the island of unlimited supplies of refined carbohydrate food resulted in a dental problem for the first time in its history; the teeth of the islanders were transformed, in a short time, from a state of positive health to a state of widespread and uncontrolled disease[9].'

177. Diet may contribute significantly to caries freedom by removing the carbohydrate substrate or base for carious attack. Caries resistance is a different matter. It is possible that other trace elements, e.g., molybdenum, may be shown to contribute to resistance and there is evidence that some phosphates, e.g., calcium sucrose phosphate[13], may also help, and as food additives help significantly, but no element or nutrient has proved to have anything like the effectiveness of fluoride. In particular, in answer to some suggestions, I quote 'there is no evidence that the high consumption of calcium-rich dairy products and of vitamin D supplement in the Western World has reduced dental caries[12]'. In the present state of knowledge it does not seem to be putting it too highly to say that fluoride is the only element proved to be significantly and generally effective in the promotion of host resistance as distinct from an artificial freedom which can only be gained by a measure of self-denial seemingly beyond the capacity of society to achieve except in the case of the young confined in institutions who may be thought to have no choice in the matter.

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CHAPTER 12. MEASUREMENT OF CARIES EXPERIENCE

Section (a)—The Diagnosis of Caries and Some Factors Affecting Estimates of Its Prevalence and Incidence

178. The clinical diagnosis of caries is a professional matter outside the scope of this Report. The measurement of caries experience in any given population is a matter of some importance and for comparisons with other populations to be valid it is necessary that there should be common standards of diagnostic methods, of description and of evaluation. When comparisons are made variables such as age, sex, diet, and living standards have to be taken into account so that the factors which influence the prevalence of caries should as far as possible be present to a similar extent in each group studied. This is not always easy to achieve[1].

179. As to diagnosis, it seems to be agreed that with ordinary methods of clinical diagnosis, i.e., mirror and probe, it is impossible to detect caries reliably until the process has reached a relatively advanced stage, and it is conceded that the results are difficult to standardise as between different observers and as between different trials[2]. Dean's criteria[3] may be taken as typical of the earlier mass surveys though terminological differences are sometimes apparent between different observers[4, 5] and these may reflect variable standards of diagnosis.

180. Methods used in later surveys have greatly refined the rather crude data of early work. They involve the use of X-rays, particularly for interproximal surfaces in which lesions may not be accessible to the probe[6], a more rigorous classification of early lesions and a detailed differentiation and classification according to site[7].

181. As to age, in the case of children, one important variable will be represented by the number of erupted teeth and whether they are deciduous or permanent. There is not much point in comparing the state of the teeth of a five-year-old with that of a fifteen-year-old. Once the permanent dentition is complete, there seems to be agreement that the prevalence of caries (which is a cumulative score) increases with age. The rate of increase tends to slow down from about the age of twenty years onwards, but in Australia at least, where no precise epidemiological study of the matter has been attempted, the reasons are not clear.

182. One possible explanation for the apparent reduction in the rate of dental decay after age twenty years might be that, when the prevalence of caries in a population is high, most of the susceptible areas of the teeth are already decayed or filled by that age; and that the only surfaces of the teeth which are not affected are those which are least susceptible. Another explanation may be that there is an increased resistance of the teeth due to some maturation process of the enamel surfaces. There are considerable variations in susceptibility to caries between different teeth, between different surfaces of teeth, and even between different surfaces of the same teeth. Thus the grouping together for statistical purposes of different caries forms may mislead[8, 9, 11].

183. In any survey of a given population the greater the particularity with which these factors are observed, the more accurate, naturally, the results. The growing recognition of the relatively crude nature of the indices used to express the results of the early surveys has led to their increasing elaboration culminating, at least for the moment, in the refined techniques of the Netherlands studies conducted by Backer Dirks *et al*[10, 8].

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Section (b)—The DMF and def Indices of Caries Experience

184. The object of all these surveys has been the same, viz., to measure and express the caries experience of a selected group. This is done by means of a ' DMF ' index for permanent teeth (indicating those which are decayed, missing or filled) and ' def ' for deciduous. The difference between the two measures is that ' e ' meaning extracted, is used to indicate deciduous teeth due to be extracted on account of caries but to exclude loss due to natural exfoliation. Each individual in the group is examined and his DMF score (or def in the appropriate case) is determined by totalling the mutually exclusive number of filled teeth, the number with untreated caries (irrespective of the number of lesions or surfaces involved) and the number either extracted or due for extraction on account of caries. Each tooth is counted once only and wisdom teeth are excluded. Thus the maximum score would be 28. This index may be expressed as a score per person, per 100 persons or 100 teeth. In later and more sophisticated studies it is more frequently expressed in terms not necesarily of teeth but of surfaces and types of surfaces, in which case molars and premolars each would have a possible maximum score of 5[1, 19].

185. In the case of young children, the problem of mixed dentition (i.e., both permanent and deciduous teeth) and of unerupted teeth can introduce further complication and to get an accurate index it may be necessary to express the score as a percentage of erupted teeth or surfaces. Thus a child of ten and a child of fourteen may have an identical DMF count but the percentage index will be much higher in the ten-year-old because of the fewer erupted teeth.

186. In children, who as a class lose relatively few teeth from disease other than caries, such as periodontal disease, the DMF index is used as a measure of the toll of caries. In older adults who lose an increasing number of teeth because of periodontal disease, DMF rates may lose their specific character in relation to caries and become instead a summary of the whole dental state, without reference to specific causes. Such figures would not have the same value for a fluoridation survey as those in which the relationship to caries is more specific. It would, of course, be difficult to be dogmatic about the precise age at which this change in character of the index becomes significant. Most authorities agree, however, in using the DMF index for measuring carious experience up to the age of twenty-five at least[1]. However, because of this and other factors, Dean advocates 12–14-year-old children as the age group of choice[2].

187. These indices are in universal use for two purposes. Firstly to indicate differences between the same group or groups of subjects in successive years and secondly as between unrelated groups. The validity of either comparison will depend on the measure of standardisation achieved but obviously the risk of introducing uncontrolled variables is greater in the second.

188. One obvious defect of such indices is that they are measures of prevalence, not of severity. Thus in a DMF survey a pinpoint lesion in a tooth would count as one; the same score as a totally decayed tooth requiring extraction, or a better example is that of a natural fissure in a six-year molar filled prophylactically as is common practice (i.e., not because of the presence of caries but because such sites are caries prone). This would achieve the same score in the index as a grossly decayed incisor whereas the clinical summation would be entirely different. Even as measures of prevalence a tooth with one lesion will be given the same score as another with multiple lesions, and if it be sought to avoid this by counting surfaces it still remains open to the comment that it will not reflect fresh lesions in and around already decayed sites.

189. The statement that the DMF index is inadequate to reflect severity apart from mere prevalence must be qualified perhaps to the extent that a substantial difference in the ' Missing ' index may, all other things being equal, such as the degre of conservative dental treatment available, reflect more extractions and hence in some measure a generally greater severity of affliction. The same point (i.e., the one made in the preceding paragraph) was made by Sir Arthur Amies in his submission but for a different purpose. As he said, ' one cannot equate a missing tooth with a well-filled vital tooth remaining for years as a functional unit of the dental arch '[3]. This is true if one is seeking to use the index as one of dental efficiency, but it is nevertheless correct that questions of severity or degree apart, teeth of both types reflect prevalence. Some of the statistical arguments arising in the course of the evidence could possibly have been avoided had the notion been firmly grasped and adhered to that DMF rates are no more than a summation of caries experience; a history in statistical form, not an accurate index of present clinical conditions.

190. It was put to me (e.g., by Professor Storey)[4] that this tends to weight these indices against fluoridation, as clinical experience supports the view that lesions occurring in fluoridated areas are, generally speaking, smaller, advance less rapidly and are more easily controlled than the frank lesions seen in unfluoridated areas. This claim I treated with reserve until I had the opportunity of testing it by demonstration. For this purpose, among others, I attended clinical examinations made by school dental officers at three country centres in Tasmania, Beaconsfield (fluoridated), Scottsdale and Forest (unfluoridated). I also attended a similar examination at Yass in N.S.W. (fluoridated). In the case of the Tasmanian examinations, I was fortunate that the examining dental officers could draw upon a world-wide background of clinical experience and one at least had had no experience of fluoridation before[5]. Their opinion was unanimous and emphatic, that caries in a fluoridated area differed qualitatively from that which their clinical experience would have led them to expect in subjects of similar age and characteristics. I accept the evidence and am completely satisfied the claim by Professor Storey which was supported by other witnesses is a valid comment. Such a conclusion is also consistent with the hypothesis that fluoride may by remineralisation play a reparative role in respect of early carious lesions[4] and is confirmed by Backer Dirks—' . . . the speed at which cavities increase in size is less in the fluoridated area '[6].

191. On the other hand, the lack of X-ray facilities in some of the earlier studies could possibly have weighted the figures in favour of a fluoridated group in comparisons made with a non-fluoridated group. The smaller the interproximal lesion the greater the chance it may escape detection by mirror and probe. Thus in one study (Evanston) Blayney and Hill[7] have reported that as a result of radiological procedures they had detected a greater percentage of proximal lesions in the fluoridated areas, though when their overall figures are studied, it will be seen that the total number of such lesions detected by all means per 100 children, was much greater in the non-fluoridated than in the fluoridated area[7].

192. It is obvious from even this cursory examination of some of the factors involved, that the value of these indices as an epidemiological method of recording differences in caries experience between different groups or ' populations ' or within the same group at different times and levels of exposure may vary. Obviously their value is highest when they are used for intra group (this does not preclude a control subgroup within the group observed) comparison by, if possible, the same observer or the same group of observers using rigidly standardised methods. Their value must lessen when used for inter group comparison, but again it will reflect the care used in the collection, analysis and differentiation of the raw statistical data, the comparability of the groups, both in dental respects and in respects other than dental and a host of other factors. This is not to say they can have no value; they may have a great deal, it is only meant to convey that there are difficulties of data collection and interpretation inherent in the problem that can never be completely overcome, though some will succeed better than others. This has been recognised, not overlooked as some critics would seem to imply[8, 9, 10, 11] and the elaboration of method and ordered inference that experience has brought[18] does not imply past failure, only differences in value.

193. In this regard while a great deal of criticism has been levelled at some of the earlier statistical studies in which these indices have been used[12, 13, 14] the meticulous care in data preparation and analysis reflected in the Netherlands Tiel-Culemborg fluoride trials I have nowhere seen criticised. In view of the controversy that has arisen over data derived statistically from the use of these indices in other trials, particularly in the U.S.A., the comparative silence over the Tiel-Culemborg (the initial data of which has been available since 1961[15]) is a matter telling strongly in its favour.

194. However this is not to discard earlier studies. That they still have considerable value is I think shown by a very useful paper referred to by the South African Commission[16] in their Report. This was a paper by Bransby and Parfit (1952)[17] who studied the relationship between the caries indices in common use by comparing the data collected in four studies by different workers. Their method of study was to recalculate on a common basis the various tables in each study. Their results showed that although these tables, employing various indices, did not provide the same degree of information, a close association in ultimate result, as measured by correlation coefficients, existed between each of them.

195. The South African Commission examined the matter closely; their conclusion was a cautious one which I would with respect adopt and which I have endeavoured to apply viz.:—

> ' Since different indices have been used to test the efficacy of various methods of reducing dental decay, we have therefore evaluated the evidence given verbally or quoted, as trends rather than as mathematically-accurate statements of fact '[16].

The only comment I would make, inspired again by the seemingly endless semantic difficulties that attend this controversy, is that, while 'trends' indicate something less than absolute proof, as evidence, they may still possess cogency, i.e., compelling force in arriving at a conclusion. But ultimately in any process of judgment (a process unnecessary in the presence of mathematical demonstration) it is the degree of satisfaction that the totality of the available evidence brings, not the semantic niceties of the labels attached to its constituent parts. I will return to this matter at a later stage.

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CHAPTER 13. CARIES AS A HEALTH PROBLEM

Section (a)—Its Prevalence

196. That caries—gross in degree—is prevalent in the Tasmanian community was agreed by all. I had at one time thought, in view of this substantial agreement by all qualified to speak, that it might not be necessary to attempt to assess the degree of prevalence. This is not easy to do in view of the lack of exact data for the State. However, it soon became apparent that even if I was satisfied that substantial benefits to dental health could be expected from the fluoridation of drinking water it would also be necessary ultimately to decide a competing matter of social principle, viz., whether a procedure which affected all alike, whether in need of it or not, was justified as being in the public interest. Such a conflict should not be resolved without information as to the prevalence of the evil sought to be remedied, the gravity of its effects, and the efficacy of alternatives. The first two of these matters I seek to examine in this chapter.

197. Caries is said to be a disease of childhood. More exact studies in America have suggested that the greatest intensity of the caries process is from 15 to 25 years of age[1]. Though these are American studies I see no reason to doubt their general validity under Australian conditions. Fifty per cent of the population of this State are 25 years of age or under, 42% are 20 or under and 20% are 12 or under[2]. The potential prevalence of this most widespread of all diseases is therefore substantial in terms of the population likely to be affected.

198. Such surveys and evidence as are available in respect of Tasmania are not as complete or informative as those that exist for other parts of Australia. However, they support without exception the generally held view that conditions among Tasmanian children are certainly no better and in all probability worse than in other Australian States. I myself share that opinion as a result of this hearing.

199. In 1954-55 Barnard[3] surveyed 7000 N.S.W school children between the ages of 6 and 15 years. Specifically he found caries of the permanent teeth present in 89.7% and of permanent or deciduous teeth in 98.5% of the children examined. It follows that less than 2% of the children examined would have been caries free and as those included three groups of children with special environments which were considered to have noticeably influenced their caries experience (two institutions with special dietary regimes and the other a naturally fluoridated towship) the general figure for the rest of the population would have been less. The caries experience for permanent teeth expressed as DMF was 6.6 per child. There were two other findings of significance, viz., malocclusion of some degree in 80% and idiopathic enamel defects in 30% of the children examined. Another 1962 survey in the same State of a sample of 12,000 primary school children showed that each child had an average of four defective teeth which needed treatment[4]. This of course is not a total DMF or def figure and should not be confused with one.

200. In 1964 the Council of the Dental Hospital of Melbourne showed its concern at 'the high incidence of dental disease' in Victoria (which they said was not substantially different from other States in the Commonwealth) by a public statement[5] designed I gather to arouse awareness of the magnitude of the problem with which current and projected resources in dental manpower in that State were inadequate to cope. Their statement included Table VII. The reference to Tasmania and its relation to comparable figures for other States will be noted.

TABLE VII

DENTAL HEALTH IN THE AUSTRALIAN COMMUNITY

		Mean DMF
Children (Aged 9)	Sydney	4.82
	Tasmania	6.0
	Western Australia	4.6
	Melbourne	4.13
Children (Aged 6-16)	Decayed Permanent Teeth	89.7%
	Malocclusion	80.0%
	Inflammation of Gingivae	75.0%
Male Adults (CMF Aged 21 years)	Teeth Missing per Mouth	7.6
	Teeth Requiring Treatment per Mouth	7.2
	Number Wearing Dentures	25.4%
	Number Requiring Periodontal Treatment	44.0%

Estimated Number of Decayed Teeth in the Community — 40,000,000

201. A Tasmanian survey[6] of 1722 children in Huon and Channel district was carried out in 1962 by Dr Brothers. Tables VIII and IX taken from this survey speak for themselves. Even more striking is Table X taken from the same study showing caries prevalence approaching 100% by the age of 13 in all eight of the 6-year and 12-year permanent molars of the children examined. It also indicates severity in that in children in the 13 to 16 age group half of all the 6-year molars had been extracted and of those left standing 52.5% were due to be extracted at the time of examination.

202. These tables reflect a survey carried out in one Southern district only. There are no other comparable surveys for other parts of the State but I received a great deal of clinical evidence tending to show that conditions elsewhere at least approach, if they do not parallel those in the district referred to. I was fortunate in this evidence as the witnesses, all of whom were trained and had practised elsewhere than in this State, were able to draw upon professional experience in many parts of the world, including the United Kingdom, Canada, and America, as well as other Australian States. This made their comments all the more illuminating.

203. I think their evidence should be summarized in order to dispel any suggestion that the picture has been overdrawn in order more effectively to promote an unpopular remedy.

TABLE VIII

PREVALENCE OF DENTAL CARIES IN THE SECONDARY DENTITION, IN AGE GROUPS 6-16 YEARS, EXPRESSED AS A PERCENTAGE OF ERUPTED TEETH AND AS A PERCENTAGE OF CHILDREN EXAMINED

School	D.M.F. per 100 erupted teeth			D.M.F. per 100 children		
	Group 1 Age 6-8	Group 2 Age 9-12	Group 3 Age 13 and over	Group 1 Age 6-8	Group 2 Age 9-12	Group 3 Age 13 and over
Kingston†	28.9	30.4	..	194	523	..
Sandfly†	26.0	32.0	..	206	587	..
Margate†	32.5	43.8	..	288	840	..
Snug	35.7	40.0	58.8	261	827	1,525
Woodbridge	36.9	41.2	62.1	312	887	1,704
Cygnet 1	33.7	40.0	52.0	273	750	1,444
Cygnet 2	37.5	46.4	64.0	300	966	1,694
Huonville	35.9	43.8	51.9	292	857	1,550
Mean	33.4	39.9	57.7	265.7	779.6	1,583.4

†Group 3 not represented.

TABLE IX

THE DENTAL CARIES INDICES OF TASMANIAN SCHOOL CHILDREN AGED 6-15 YEARS AND THOSE OF CHILDREN FROM THE UNITED STATES OF AMERICA, NEW ZEALAND, CANADA AND NEW SOUTH WALES

Age in years	Tasmania		Mean D.M.F. per child							
	No. of children	Mean D.M.F. per 100 teeth	Tasmania	U.S.A.		New Zealand		Canada	New South Wales	
				Grand Rapids	Muskagon	1	2	Brantford	U.D.H. Sydney	State Schools
6	108	31.5	1.5 (1.5)*	..	..	1.41	..	..	2.05	0.99
7	134	33.9	3.0 (1.3)	1.89	1.99	2.75	2.01	1.87	2.94	2.31
8	160	36.9	4.3 (1.2)	2.95	2.81	3.73	3.09	2.40	4.09	3.22
9	137	39.3	6.0 (2.4)	3.90	3.81	4.45	3.82	3.87	4.76	4.44
10	139	39.7	7.4 (3.9)	4.92	4.91	5.48	4.44	3.71	5.82	5.28
11	104	43.4	9.6 (3.7)	6.41	6.32	7.12	5.62	4.60	8.27	6.98
12	88	50.5	12.0 (3.5)	8.07	8.66	9.47	7.08	6.41	..	9.32
13	77	54.0	14.7 (3.8)	9.73	9.98	11.82	8.87	7.61	..	10.70
14	65	60.5	16.3 (4.2)	10.95	12.00	14.35	10.01	8.75	..	12.78
15	22	59.7	16.4 (3.1)	..	..	16.80	..	..	..	13.91

*Standard deviation in brackets.

TABLE X

CARIOUS FIRST AND SECOND PERMANENT MOLARS IN CHILDREN AGED 6-16 YEARS

School	First permanent molars									Second permanent molars	
	D.M.F. /100 erupted			M /100 erupted			X /100 erupted*			D.M.F. /100 erupted	
	Group**			Group**			Group**			Group**	
	1	2	3	1	2	3	1	2	3	2	3
Kingston†	64.0	97.0	..	2.2	14.0	..	4.1	13.2	..	50.0	..
Sandfly†	55.5	95.0	..	0.0	17.0	..	10.0	50.0	..	No data	..
Margate†	81.5	99.0	..	1.7	22.2	..	16.6	33.0	..	48.0	..
Snug	77.7	97.5	100.0	1.3	17.7	50.0	12.3	46.0	52.5	77.3	93.3
Woodbridge	80.0	100.0	100.0	2.8	17.6	51.0	6.6	48.0	50.0	79.0	97.0
Cygnet 1	78.6	97.5	100.0	1.2	18.7	42.7	7.8	32.7	41.7	49.0	77.0
Cygnet 2	86.3	99.7	100.0	4.4	25.0	57.1	11.5	50.0	76.6	66.0	95.0
Huonville	82.3	97.9	100.0	1.8	16.0	49.8	10.0	31.6	41.7	58.5	94.0
Mean	75.8	97.9	100.0	1.9	18.5	50.1	9.8	38.0	52.5	61.1	91.3

* The Author has since advised that 'erupted' should here be read as "standing". It is so treated in the text.
** For age distribution see Table VIII supra.
†Group 3 not represented.
Taken from P. C. Brothers[6]

(1) Dr Ramsden[7] had qualified and practised in England. He characterised the dental health of the school population of the Scottsdale District in which he had served for two years as a School Dental Officer as very bad. In his first year of practice in a school population of 1800 he extracted 3250 teeth and did 2200 fillings. In many children in the 6, 7, and 8 year group the deciduous molars were so badly decayed that 'there was nothing to do but extract them. I was extracting eight (i.e., all) deciduous molars for numerous children'. As against his experience in England where to supply a child under 18 with full dentures would be an outstanding thing (in fact in 10 years of practice he could not remember a single case in a population of 17,000), he found it commonplace, as did other dental surgeons in Tasmania and he had had to do clearances of either or both jaws for 21 school children in his first year.

(2) Dr Eslake of Smithton had qualified and practised in Sydney. He had also practised for 13 years in Great Britain, in the East End of London. Like Dr Ramsden he had not previously experienced the necessity for full clearances in children under 18 until he came to Tasmania. The rate at which children's teeth decayed in Tasmania was 'much higher' than anything he had experienced before. He found many children with carious lower front teeth. 'I can't ever remember anything like the number of children or adults with carious front teeth that I have seen in Smithton. This used to be regarded as being a rarity . . . the beginning of the end . . . They (the lower front teeth) seemed to be immune in normal areas. As far as I am concerned, I can regularly find them in the children with teeth that are almost too far gone to adequately fill, in the lower anterior region of the mouth'[8]. He had had to extract 6-year molars, 12- year molars and bicuspids before the root was formed, a thing he never remembered having to do before. He also spoke of frequent clearances and speaking generally of children and adults he said 'people are not dentally conscious, and you cannot expect them to be when their dental state is so poor as it is. Most of them expect to have false teeth before they are married. I quite regularly get girls and boys who come in and see me in their late teens, early twenties—"I don't want any fillings done at the moment. Just take this one out because it is aching—because I am going to get them all out next year—because I'm being married". This is almost standard procedure; it is almost part of the dowry. They have not any future as far as keeping their teeth is concerned. The state of their teeth is so poor that you just are putting your finger in the hole in the dyke trying to patch them up until they are a bit older and they will have them out'[8].

(3) Dr Arkle, the senior School Dental Officer for Tasmania since 1962, had practised in Scotland and Canada, including Labrador and Newfoundland. He had had a wide experience of Tasmanian school children. He confirmed the decay of lower anteriors as an abnormal feature of the Tasmanian carious picture particularly in the Midlands and in fact had made a special study of the matter, drawing on the school records from all districts. In relation to these teeth he had found the Tasmanian position so remarkable dentally that he had drawn it to the attention of overseas authorities who had confirmed it as unusual and as indicating a highly active caries situation[9]. In sum his evidence established the prevalence of this serious clinical portent, occurring in over 20% of Tasmanian school children, as probably the highest reported anywhere else in the world[10, 11, 12, 13, 14].

(4) Dr Stabler, referring to his experience as Dental Surgeon at the Launceston General Hospital before going to Beaconsfield as School Dental Officer, confirmed the high rate of total clearances necessary in young Tasmanians under 20. After referring to a case of a girl of 14 years who needed a full clearance of both jaws, he was asked:—

> '*What would be the incidence of that sort of example at the Launceston General Hospital?*' . . . 'Oh heavens, we had two sessions weekly generally in these cases, at which on an average we would see about eight patients each session, and most of these people would be under twenty, having full clearances.
>
> '*From how young?*' . . . 'From eleven upwards'.

'Just give me those figures again?' . . . 'We would have two sessions weekly, and we would see about eight patients each session, this is for general anaesthetics, and I would say the majority of those people would be under twenty' [15].

204. Figures of clearances in school children at the Royal Hobart Hospital were not available. However, in addition to its routine clinical services and subject to need, the School Dental Service provides free dentures to children aged 12 to 16 years. During the period February 1966 to June 1967 it supplied five children (including a girl of 12) with full upper and lower dentures, 25 children with a full upper set and one child with a full lower set. Partial dentures supplied in the same period numbered 88. The average age was 14 years[16].

205. I had other evidence from doctors as well as dentists to the same general effect. Admitting the danger of relying on clinical recollection and impressions, I sought other ways of testing the matter. The Department of the Army seemed a likely source and Dr Brothers, as D.A.D.D.S., Tasmania Command, gave evidence to the following effect.

206. A survey (1956-57) of the last intake of 1215 17-year-old Tasmanian National Servicemen gave the following results[17]. Only 9% were classified as dentally fit following what was described as a 'relatively superficial' dental examination. Three hundred and one (or 25%) were wearing some form of denture and an approximately similar number were in need of one. Five hundred and fourteen required extractions of up to four teeth and 175 required more than four. At least 5040 major cavities required filling. The percentage of Tasmanians requiring extractions and wearing or requiring dentures was considerably higher than the average Australian figure or for any other Australian State. I take the following figures from his evidence:—

Percentage of National Servicemen (17-year-old)—

(*a*) Wearing dentures	N.S.W.	15%
	Australia	12%
	Tasmania	25%
(*b*) Requiring full dentures	N.S.W.	2.3%
	Australia	3.4%
	Tasmania	7%
(*c*) Requiring partial dentures	N.S.W.	22.4%
	Australia	22.8%
	Tasmania	23.5%
(*d*) Either wearing or requiring some form of dentures	N.S.W.	40%
	Australia	42%
	Tasmania	54%

207. These figures may be compared with those for the 1965 intakes of National Servicemen (21-year-olds)[18].

		First Intake	Second Intake
Percentage unfit	N.S.W.	91%	82%
	Australia	91%	86%
	Tasmania	94%	86%
No. of teeth requiring filling	N.S.W.	4.1	3.0
	Australia	4.2	3.4
	Tasmania	4.4	3.3
No. of extractions required per person	N.S.W.	0.8	0.6
	Australia	1.1	0.8
	Tasmania	1.9	1.9
Ratio of recruits requiring dentures	N.S.W.	1 in 17	
	Australia (not available).		
	Tasmania	1 in 3	

The last figures (dentures needed) do not include those already wearing a satisfactory denture at the time which could entitle them to be classified as fit if no other treatment were needed.

208. Other figures were made available to me but I see no point in prolonging the story. As a summary estimate it can be said that the results consistently indicate that approximately 40% of young male Tasmanians of National Service age either wear or need at least one full denture. It could be more. It is little wonder that one witness referred to the caries prevalence in children of this State as being higher 'than in any comparable survey of white children that the author has been able to find published'[6].

209. I have asked myself whether it would be possible to convey by photographic means something of the carious picture that I have seen in the course of attending at examinations made by school dentists in various parts of the State. Photographs were taken as a matter of routine in the hope that it might ultimately be possible fairly to reproduce something of what these examinations revealed as to the prevalence of caries and its degree. I say fairly because merely to reproduce them as examples of carious mouths seen would really mean little and they could by their emotional appeal actually mislead. Unless ordered in some form of epidemiological sequence they would be nothing but examples of pathological specimens leading to no valid inference. Unfortunately the data available does not enable this to be done. Conversely, the same reasons exist for not using other photographs of caries-free or relatively free mouths seen, for example, at Beaconsfield or Yass. There are great difficulties in arranging any selection of either kind and avoiding a charge of a weighted or an invalid comparison. I am not able to portray on any epidemiological basis of relative frequency what is the typical carious mouth or the reverse. Further, many photographs, even of seemingly good mouths, can mislead unless accompanied by a full clinical description of details which the camera may not and often does not reveal. For these reasons I reproduce none.

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Section (b)—Its Effect

210. There is no exact method of measuring the effect of caries on the health and well-being of the community until perhaps one arrives at the terminal state involving in the individual the complete loss of the dentition in one or both jaws. For this reason, in the preceding section and in what follows, I have tended to attach some importance to the age at which this state may be reached by the young people of the community.

211. Simple caries may cause pain particularly in children. I have no means of estimating its extent or degree. Simple enamel or dentinal lesions, although they mar the physical integrity of the body, do not, generally speaking, impair in any material way the general health or physical well-being of the subject affected. Neglected or advanced caries will eventually lead to tooth loss either by extensive destruction of the hard dental tissue or by septic involvement of the pulp and via the pulp of the bone and tissues surrounding the root[1]. In this manner inefective processes are brought into contact with the blood stream permitting transport of bacteria and toxins to other parts of the body. This has been demonstrated by positive cultures for harmful organisms being obtained in the blood of patients who have had extractions, whereas they were

negative before the operation[2]. One witness likened the effect of masticatory pressures on an apical abscess to the force-pumping of deleterious material into the vascular system[2].

212. Though the same importance is not now attributed to the general idea of carious teeth acting as a septic focus for the spread of toxic organisms and products throughout the body as formerly, all witnesses before me agreed that the notion could not be discarded. Specific disorders which I was told can be associated with oral infections are tonsillitis, sinusitis, other infections of the mouth and adjoining structures, respiratory disorders of childhood from inhaled pus and infective material[2] and, from blood-borne infections, infective endocarditis which, if it occurs, will either be fatal or seriously impair health for the rest of the child's life. Such cases are uncommon but they occur as do fatalities under anaesthesia administered in the course of dental surgery[3].

213. In general, however, even in the case of advanced caries, a definite systemic effect on the health of the sufferer is hard to demonstrate. It is not a direct cause of mortality, nor even of recognizable morbidity. As one writer has put it, it belongs to that class of disorder which, while painful and distressing, does not disable or cripple 'and its victims may be, for the most part, those who are seldom ill but never well; those who "enjoy" poor health and yet may live to a ripe old age'[4, 2].

214. Caries is progressive and once the lesion is established for all practical purposes it is irreversible[5]. Apart from the remineralization of incipient lesions (Ch. 11 (c), *supra*) there is no natural regenerative process whereby lost tooth substance is or can be restored. The only way to repair the damage is to remove the diseased part by mechanical means and to restore the lost substance artificially. Ultimately, if unchecked, caries leads to the loss of the tooth itself by extraction. Premature extraction of deciduous teeth due to caries is a common cause of orthodontic disorders such as malocclusion. These can result in an altered appearance due to interference with the natural development of the jaw and facial features. But loss of any teeth brings in its train problems of functional efficiency. Loss of masticatory ability often results in an unconscious modification of the diet to favour those substances mainly cariogenic which do not require chewing. In the young particularly this change in diet makes it more likely that the teeth will be attacked by caries. At all ages it may change dietary habits and effect nutritional status.

215. When the final stage is reached of a 'clearance' of one or both jaws, there is extensive consequent loss by resorption of alveolar tissue and jaw bone substance[6] and subtle effects in relation to organs of taste, hearing, and speech[7].

216. Dentures should be renewed regularly to accommodate to the resorption of underlying bone and tissue in the dental arch. This is seldom done as frequently as it should be and ill-fitting and dirty dentures are a commonplace. There is a clinical correlation recognized between ill-fitting dentures and malignant conditions of the oral cavity which may follow leukoplakia[3] though a direct causal relationship has not been demonstrated. For a few persons with lip, jaw and palate defects the complete loss of teeth in either or both jaws can be a very serious matter as it may prevent or interfere with surgical restorations and protheses[7]. Though these are but a small section of the community, it is to be remembered that in relation to hare lip and cleft palate Tasmania had in 1960 the highest recorded prevalence (1 in 600 births) for a community of European stock[8].

217. It seems clear that the viewpoints of the dental profession and the community on the subject of dentures are not the same. Dentists who know more about these matters and their generally unperceived but sometimes far-reaching effects tend to become emotional in their despair:

> 'The average Australian while apparently physically and mentally fit, is a dental cripple. His oral apparatus relies for its function on crutches of amalgam restorations, synthetic resin dentures and porcelain teeth. Although by now he understands that sticky lollies produce dental decay he ignores this and buys more than 120 lb of sugar a year which he consumes largely between meals, at the wrong time. In this way, he contributes to the more than 40,000,000 decayed teeth in the community. He buys one toothbrush a year instead of the three recommended by the dentist and uses it once a day, usually at the wrong time and in the wrong way, so that by and large it does more harm than good. His visits to the dentist are infrequent, and are largely prompted by pain and discomfort where, sensing that dental treatment alone does not stop the progression of dental disorders he insists that his teeth be removed and artificial dentures inserted. He consoles himself with the thought that Australians are predestined to have the worst teeth in the world and does not want to understand that all civilized countries are experiencing the same problem'[9].

On the other hand the community attitude to the edentulous state appears to be one of resignation and acceptance, sometimes even relief. Unless one can believe that the teeth like the vermiform appendix are but evolutionary vestiges it is hard to understand evidence, which I accept, as to the commonly expressed desire of young Tasmanian girls to have total clearances before marriage so that their teeth would not give trouble thereafter. As one dentist put it it tends to be regarded as part of her dowry.

218. A comment on the prevailing community view was made by Professor Ewing, Professor of Surgery at Melbourne University, in the 1963 Stawell Oration:

> 'Dental caries tends to be regarded as no more than a by-product of our times, an inescapable retribution for dissipation in the sweet shop and in the milk bar. Even if we accept that the edentulous state is not socially so allowable as, let us say, baldness, the young man, or even the young woman, so afflicted suffers no stigma, always provided he can afford to cover up his deficiency with a row of miserable substitutes. He is widely believed to be in no way handicapped by the absence of the real thing; but we know very well that a man in early life with a row of healthy teeth has the literal, as well as the metaphorical, edge on his unfortunate, toothless fellow citizen'[10];

but the sting is in the last sentence.

219. I cannot measure these things in terms of money or economics or national wealth or industrial output or even educational loss. I note that the State possesses a School Dental Service on which it is spending a sum in excess of $300,000[11, 12] a year and would probably spend more were the skilled personnel available for its needs. I note too this service is 'flooded'[13] to the extent that for the Hobart area it has to restrict its prophylactic services to children of eight years and under; older children being given emergency treatment only for the relief of pain or for some other urgent dental problem such as clearances and prosthetic appliances[14, 11]. Unfortunately, for the population as a whole it seems quite clear that the dental manpower of the State is insufficient for the State's needs and that the present Tasmanian ratio (already the lowest in the Commonwealth)[15, 16] of one dentist or technician to approximately four thousand of the population as against an optimum of one to 1700[16] and a 1963 all-Australian figure of 1 to 2500 is unlikely to be maintained and will further decline[15].

220. I believe the position to be one that calls for serious thought. The evidence is unanimous. There is no prospect that the carious tide will be contained, let alone turned back by the available dental services of this State, present and prospective. This is the unanimous conclusion of dental professor, administrator and practitioner alike. Unless there is some major change in dietary habit it would seem probable that 50% of the population will enter adult life with one or more dentures. This, as Professor Ewing said in the Stawell Oration already referred to is not a very distressing problem in a compelling and urgent sense 'for even when all the teeth have been lost or are useless, our patient will still survive'[10]. At the same time childish pain, trauma and psychological shock and the economic and educational loss that it reflects is serious enough to be considered as a grave problem of public health.

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PART F—THE FLUORIDE HYPOTHESIS AND ITS VERIFICATION

CHAPTER 14. GENERAL AND CLINICAL

221. Some physiological and chemical mechanisms have been described from which a presumption of dental benefit from fluoridation may be drawn. That fluoride is not capable of affecting the teeth and increasing caries resistance was not strongly suggested, at least by anyone whose views would carry weight. And not all who opposed fluoridation were against the use of fluorides at least by the young. Thus Sir Arthur Amies, who is opposed to the fluoridation of water supplies, would nevertheless advocate their topical application ' in the present state of our knowledge '[1] and Sir Stanton Hicks would administer it in the expectation of dental benefit as a matter of individual prescription[2], to mention two.

222. What was suggested, was, generally speaking, that the beneficial effects were overstated, that the possibilities of unfavourable side effects had not been excluded and that the method of administration was unethical and involved a breach of Hippocratic principle. Hence I now pass to the evidence (in which of course I include the great mass of written material literally from all over the world and which, rather than the spoken word, became the common coinage of the Commission) as to how far this hypothesis of benefit has been verified by clinical observation, epidemiological investigation and experimental application.

223. Of the clinical I propose to say little. Impressions gathered in this fashion are subjective only and lack the cogency of controlled experiment and examination. All Tasmanian experience fell strictly into this category. Though some interesting figures[3] were given in relation to Beaconsfield (now fluoridated for fifteen years), they should not I think be put in any higher light than clinical records, lacking the cohesion and sustained planning of a controlled trial. However, it should be recorded that all dental practitioners and members of the medical profession who gave evidence and who had had clinical or other practical experience of the dental results of fluoride, were unanimous in their opinion of substantial benefit. In fact I found the unanimity of these opinions and the degree of conviction with which they were expressed most impressive. I was present when three dental surgeons made jointly an examination of the teeth of children attending schools in Beaconsfield. I quote as typical Dr Ramsden's evidence taken immediately the examination was concluded. Though a dentist of many years experience in both Australia and the United Kingdom it was his first introduction to fluoridation in practice[4].

> MR JENNINGS: '*Doctor, had you seen the teeth of the children of Beaconsfield before today?* . . . No.
>
> '*What do you have to say of the teeth you saw of children who had fluoride all their lives as compared with the children in Scottsdale whom you are regularly seeing?* . . . Well I was most impressed with this. I, of course, read a lot on fluoridation and keep in touch with it but I had never seen it. I am much more impressed than I thought I was going to be. It seemed to me if I didn't know otherwise, that these children had been specially selected to show me the best children you have got. I presume this is a random selection.'.
>
> MR COMMISSIONER: '*I take it the emphasis is on the "otherwise"? If you didn't know otherwise, you would have thought* . . . Yes that is right. I hope you understand my meaning.
>
> . . . *because I assure you great pains were taken not to do that.*'.
>
> MR JENNINGS: '*It being a completely random selection.* (Witness continuing): The general dental health of those children taken as a whole was very good indeed in my opinion.'.

Then there was a discussion as to the state of oral hygiene which I omit.

> ' I have one or two other points I would like to bring out. The first thing, which is something I did not expect, I wasn't prepared for, and it seems to me extremely significant, and that is, the apparent effect of fluoride on the teeth of children where they have only had the fluoride through drinking school drinking water. I did know, of course, that fluoride acts as it passes over your teeth or the fluoride in the water acts on the surfaces of the enamel as well as getting into the teeth through your bloodstream to your teeth which are being formed, but it seemed to me, from being here today, that fluoridated water just at school is such a help to the children's dental health as to be quite significant . . .'.

224. I found it odd and a matter of remark that two academic members of the dental profession, who gave evidence in opposition to fluoridation and who have identified themselves with the opposition case over the years, not only had no clinical experience of the results of fluoridation but had not sought any, though in my considered opinion had they desired it, it would have been easily obtained. Indeed my impression was, after questioning them on it, that they were not really interested in obtaining it. However, this is a matter that reflects more on the witness than his evidence. I draw no definite conclusions from it. I found it puzzling and still do, but perhaps one can get tired of controversy in dentistry as in other fields. However, while clinical impressions are not without weight, they lack the cogency of more formal and disciplined studies.

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CHAPTER 15. EPIDEMIOLOGICAL DATA FROM AREAS OF NATURAL FLUORIDATION

Section (a)—Historical

225. I make no apologies for glancing at the history of the matter. I think it is important not only as providing an informed background to the dispute which developed, a dispute in which rancour has been allowed to outstrip scientific discretion but as showing also the extent and the wealth of the clinical and epidemiological studies which preceded the statistical argument which has, in the minds of many, become the main issue. Such a view is misconceived as the history of this much researched subject shows. Giving the matter a perspective in time one may distinguish three separate though overlapping phases, of clinical observation leading to epidemiological survey and finally to experimental application. In the first two we are concerned with naturally fluoridated water, in the third with its artificial simulation. The statistical controversy initiated by Dr Sutton belongs to the third, though there are criticisms in relation to the second also to be considered. Concurrently with all three there have, of course, been chemical, biochemical and physiological studies of all kinds, *in vivo* and *in vitro*, the details and content of which defy summation in a total bibliography which may even now be approaching the 20,000 mark.

226. In the nineteenth century there had been sporadic suggestions of an association between fluorine and freedom from caries, but the modern story begins in the first decade of this century with the investigations of an American dentist, Frederick S. McKay, who set out to find the cause of the mottled discoloured teeth, so common among the population of Colorado Springs, a city in the United States of America. The story of his patient and prolonged search spread over twenty-two years is told in very readable form by D. R. McNeil in 'The Fight for Fluoridation'[1] for which reference I am indebted to Professor Polya.

227. An early phase of his investigation was to determine the geographical prevalence of this condition and to this end McKay traversed the length and breadth of the United States, and extended his inquiry to other parts of the world as well. In 1916 in collaboration with G. V. Black, Dean of the Northwestern University Dental School, he published a study of mottled enamel in which he showed that the condition was limited to well defined geographical areas and affected only those who were born and lived in the area during the years of tooth formation. He advanced the hypothesis that the drinking waters 'contain something that is not commonly detected in the standard water analyses'. At the same time he commented on what at the time seemed a paradoxical phenomenon that despite the obvious defect of severe mottling, such teeth were no more susceptible to caries than other teeth not so affected. This observation was repeated over the years and confirmed by others in the United States and elsewhere[2].

228. The elusive cause (fluoride) was first identified in 1931 following an investigation into endemic mottling at a place called Bauxite where, by employing analytical methods a little in advance of those generally employed at the time, it was found that the water supply contained 13.7 ppm of fluoride. Analyses of drinking waters in other endemic areas quickly followed and gave confirmatory results, all waters tested showing 2 to 13 ppm of fluoride[3].

229. Concurrently, but apparently unknown to McKay, a husband and wife team, H. V. and M. C. Smith[4], at the University of Arizona experimenting with rats found that high concentrations of fluoride added to their diets caused mottling, thus adding independent experimental proof to McKay's epidemiological inferences. This was confirmed by Elvove[5] in America (1933) and Ainsworth[6] in England (1933).

230. The association between mottled enamel and fluorine being thus established, the United States Department of Health assigned H. Trendley Dean to its further investigation. Between 1931 and 1938, when the significant paper was published, there were many reports both in America and from other countries suggesting a relationship between mottling and freedom from caries, and this naturally led to the inquiry whether the same agent, fluoride, might not be responsible for both factors and if so what level or concentration was necessary to produce either or both effects. In 1938 Dean[7] pointed out that there was an inverse relationship between endemic mottling and caries prevalence but the pinch of the question obviously was whether the caries protective factor was dependent upon the presence of mottled enamel or not. His investigations showed that it was not. To quote:

> 'This relative freedom from dental caries was present in deciduous teeth as well as in the permanent teeth, and *whether or not the child's teeth showed macroscopic evidence of mottled enamel*'[8].

The D.M.F. index was not then in use and the basis of measurement was as between totally caries-free mouths or otherwise. But it was sufficient to show a broad but striking correlation:

TABLE XI

NUMBER AND PERCENTAGE OF CARIES-FREE NINE-YEAR-OLD CHILDREN OF SIX SELECTED CITIES

(Classified on the Basis of Continuous Exposure to Domestic Water of Lower and High Fluoride Concentration)

Fluoride (F) content of public water supply in parts per million	Total No. children examined	Caries-free Children					
		All teeth		Permanent		Deciduous	
		No.	%	No.	%	No.	%
0.6–1.5*	114	5	4	30	26	13	11
1.7–2.5	122	27	22	60	49	38	31

* Original report notes that water supply listed as 1.5 (East Moline, Ill.) may be subject to correction to 1.3 parts per million.

Taken from Dean (1938)[7].

231. Analysis of this and other studies indicating the same result led Dean to test the hypothesis that the caries protective factor was not dependent on the concurrent existence of mottled enamel by detailed local investigations. The first was made in respect of four cities, viz., Galesburg and Monmouth where the communal water supplies contained 1.8 and 1.7 ppm of fluoride respectively and, as controls, Macomb and Quincy, where the corresponding value in both cases was 0.2 ppm.

232. At this point the modern controversy may be said to start. The major report was published in 1939[9] but it drew upon data collected in previous years and published in other papers. Despite the obvious care taken in this stage of caries epidemiology to eliminate variables and to evolve techniques of investigation that might stand up to critical survey, in this, and in later studies, some flaws have been detected. The factors which produced them are not difficult to perceive. Comparisons and parallels are always defective in some detail or another and epidemiological studies in particular lend themselves to criticism which will be seen as pin-pricking by the worker who has tried as far as he can, to reduce to uniformity an inherently intractable subject. To the supporter, perhaps convinced already, such details will be of little consequence but they can appear as vital to the critic who does not agree and does not want to agree with the conclusion anyway. I realise of course that emotional attitudes should have no place in a scientific controversy: unhappily my experience in this matter indicates clearly the reverse.

233. The pursuit of this endless paper controversy of which this report may be said to mark the beginning, has caused me much difficulty. I could, of course, painstakingly seek to evaluate each paper and each point of detailed criticism. Such a task would in the long run still not permit of an unqualified answer, nor even a statistical one, without the aid of sophisticated techniques in which I am not versed. Alternatively I could attempt to survey this vexed sea of material in the mass; treat studies such as those to which I am about to refer as indicating trends only and not as stating conclusions and then see how those trends have been confirmed by later more rigorously collected data, on which I feel I can rely. The latter is both easier and more satisfactory; after all, it is the conclusion to which the evidence leads that is important, not who should win the debate.

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Section (b)—The Galesburg-Quincy Study

234. I will deal with this study[1] (the four cities study or as it was later known the Galesburg-Quincy Study) in some detail so as to illustrate both the nature of the controversy and the atmosphere in which it has been conducted.

235. The four cities selected fell into two groups:—

	Pop. 1930	Fluoride content of water supply in parts per million (ppm)
Galesburg	28,830	1.8
Quincy	39,241	0.2
Monmouth	8,666	1.7
Macomb	8,509	0.2

They were all in N.W. Illinois and in the planning of the survey an attempt was made to take into account all obvious variables, such as age, sex, race, diet, demographic factors and climate. Water supplies were studied and their geological sources defined. For the fluoride content of Galesburg and Monmouth waters the authors drew on a previous study[2] which reviewed the water history of Galesburg in particular and established that the supply had been substantially changed in 1928 in that prior to that date 40% of the city supply had been drawn from a different geological strata. Monmouth, on the other hand, had a continuous water history since 1926. In the case of these two cities fluoride content had been determined as the mean of twelve consecutive monthly samples taken between 1933 and 1934. This was tested against a single sample of each collected in 1938. Macomb had two sources of surface supply. Later (1946) Dean expressed himself as dissatisfied with the Macomb water data on the basis that the two sources made it impossible to determine 'with a fair degree of accuracy whether or not the chemical composition of the water used at the time of survey represented approximately the type of water used during the period of tooth calcification, ten or twelve years previously'[3]. Quincy used treated river water and the source had been in continuous use for many years. From the 1939 Report it might appear that the fluoride values for these two cities were determined on single samples, but in the 1946 review of the same work he states that they represented a mean of twelve consecutive monthly values.

236. In the original paper it was stated that the plan of study called for a comparison of Galesburg and Monmouth with Quincy and Macomb. The study was to be limited to twelve, thirteen and fourteen-year-old children born in 1924, 1925 and 1926. The children examined were divided into two groups, those with continuous exposure since birth and those who, having

been born elsewhere, had entered the community prior to the sixth birthday. Dean recognised that this meant a variable water history prior to that age but his criterion was continuous post-eruptive exposure. The importance now attached to pre-eruptive influence would mean that another major variable had been introduced, but these were early studies. He further records that attempts to give statistical significance to the water history of those children in Galesburg who had only used the city water since their sixth birthday, proved impossible. Because of this 'there was no apparent reason for making this classification at Quincy and in view of the larger number of children available, examinations in that city were, in the main, largely confined to children with a history of continuous use of the municipal water from birth'[1]. He retained however, in his calculations the Galesburg children of this group, but the basis of selection of the few from Quincy of the same classification is not explained. These factors do affect of course the strict comparability of the groups studied.

237. His findings were as stated in Table XII. The clear trend in favour of the fluoridated water supplies is confirmed by analyses of the total carious permanent teeth per 100 children examined and by another comparison (of greater interest in the light of modern views as to the importance of fluoride in inhibiting this form of caries) of caries experience in relation to the interproximal surfaces of the four superior permanent incisors.

TABLE XII

SUMMARY OF THE INCIDENCE AND AMOUNT OF DENTAL CARIES IN SELECTED 12- TO 14-YEAR-OLD WHITE CHILDREN OF 4 ILLINOIS CITIES

City	No. of children examined	Children with one or more carious permanent teeth		Children with caries-free permanent teeth		No. of carious permanent teeth per 100 children			
						Age in years last birthday			Total[1]
		No.	%	No.	%	12	13	14	
(A) 696 CHILDREN WITH HISTORY OF CONTINUOUS USE OF PUBLIC WATER SUPPLY									
Galesburg	243	155	63.8	88	36.2	177	207	201	194
Monmouth	99	63	63.6	36	36.4	115	213	271	208
Macomb	63	54	85.7	9	14.3	315	422	367	368
Quincy	291	279	95.9	12	4.1	563	615	732	628
(B) 885 CHILDREN EXAMINED INCLUDING THOSE IN (A) AND THOSE WHO HAVE CONTINUOUSLY USED THE CITY WATER SINCE 6 YEARS OF AGE; WATER HISTORY PRIOR TO 6, VARIABLE									
Galesburg	319	207	64.9	112	35.1	182	226	196	201
Monmouth	148	96	64.9	52	35.1	150	200	266	205
Macomb	112	96	85.7	16	14.3	346	411	453	401
Quincy	306	294	96.0	12	4.0	567	623	740	633

[1] Inasmuch as the age-sex distribution of the 4 samples (table 3) is relatively uniform, no attempt has been made to adjust for age-sex differences in the rate given as "total" in this and succeeding tables in this paper. This rate, therefore, expresses the amount of dental caries observed in the 12- to 14-year-old children taken as a group.

Taken from Dean[1].

238. Dean summarised his findings thus. (To assist the reader, I have inserted the letters 'F' and 'NF' to indicate fluoridated cities or otherwise.):

> 'In the 319 children examined at Galesburg(F) and the 148 examined at Monmouth(F), the number of carious permanent teeth per 100 children was 210 and 205, respectively. At Macomb(NF) and Quincy(NF), examination of 112 and 306 children disclosed rates of 401 and 633. In other words, there was approximately twice as much dental caries at Macomb(NF) and more than three times as much at Quincy(NF) as was observed at Galesburg(F) or Monmouth(F). It must also be remembered that the amount of dental caries at Quincy(NF) is not unusually high (633); it merely appears high in relation to that of Galesburg(F). . . . The differences in the percentages of children caries-free with respect to their permanent teeth is of additional interest. At Galesburg(F) and Monmouth(F) about 35% of those examined were caries-free; in Macomb(NF) and Quincy(NF), only 14 and 4%, respectively, were free from dental caries.'[1].

The point to note is that while the relationship between fluoridated and unfluoridated is unmistakeable, the difference between the two unfluoridated cities is unexplained and points to some unknown variable.

239. Of this study Dr Exner[4] has been the chief critic. In his submission to the Commission he stated:

> 'The study on which the original claim "of two-thirds reduction" in decay was based was misrepresented. Children in four towns were compared: Galesburg (1.9 ppm F), Monmouth (1.6 ppm), and Macomb and Quincy (each with 0.2 ppm). But when the data from the four towns were compared, the results were not as desired; so the data from Macomb and Monmouth were ignored and the study became known as "The Galesburg-Quincy Study". *Pub. Hlth Rep.*, 54: 862-888. May 26 1939.'.

240. The reference given is the one to which I have referred and which so far from ignoring the results from Macomb and Monmouth gives them in detail. It may be, though he does not say so, that Dr Exner has in mind the fact that in subsequent studies of other cities the figures of Galesburg and Quincy were used preferentially to the others as showing 'a striking example of a marked variation in the intensity of dental caries attack . . . the variable seemingly responsible being the difference in the fluoride content of the domestic water supply'[5, 6] or it may be that the larger sample sizes were the deciding factor but the statement that the results 'were ignored' is not supported by the reference given in the text.

241. Dr Exner's criticism continues:—

> 'Also, Dean knew before he ever undertook the study that Galesburg did not meet his own "requisites for quantitative evaluation" because of changes in the water supply during the lives of the children examined; and he had so stated in reporting a previous examination he had made there. (*Amer J. of Pub. Hlth*, 26: 567-575. June 1936).'[4].

The second comment is correct to the extent that prior to 1928 40% of the Galesburg water supply had come from a different geological strata, namely a St Peter sandstone. In a further document which found its way to the Commission, Dr Exner referred to the matter at length and to Dr Dean's evidence on the same subject—quoting from his cross-examination—in a 'fluoridation suit' in 1960 which sought (unsuccessfully) to enjoin the fluoridation of the water supply of the city of Chicago[7]. Apparently in the general area St Peter sandstone had an average fluoride content of 2.5 ppm with a range of from 1.7 to 4.0 as against Cambrian (in use since 1928) values of 1.0 to 1.9 ppm with an average of 1.5 ppm and apparently Dr Dean misquoted some of the figures in the witness box. The upshot of it all is that in Galesburg between 1924 and 1928 the 12-, 13- and 14-year-old children examined in 1938 had been exposed to a water supply 40% of which had an average content of 2.5 ppm as against the average of 1.8 ppm used in the report. This would not be of any significance in relation to the caries picture[10]. All that Dr Exner has done is to draw attention to the possibility that the water supply in question had for the years mentioned more rather than less fluoride than Dean claimed.

242. Further criticism is directed to the fact that 'there was the same percentage difference in DMF' (i.e., in caries reduction) 'between Monmouth(F) and Macomb(NF) as between Macomb(NF) and Quincy(NF)'. This may be a way of looking at it. The figures were:—

	Children examined	DMF per 100 children
Galesburg	243	194
Monmouth	99	208
Macomb	63	368
Quincy	291	628

and the percentage differences were:—

Between Galesburg and Quincy	69%
Between Monmouth and Macomb	44%
Between Quincy and Macomb	41%

It does not of course destroy the difference between Monmouth(F) and Macomb(NF) (44%) or the even greater difference as between Galesburg(F) and Quincy(NF) (69%). It might be wiser to take the size of the respective samples into account than to conclude, as Dr Exner does, that 'The differences are substantially the same; and the "benefit" that the study was alleged to show vanishes'[8].

243. Apparently in the suit to which Dr Exner refers at length Dr Dean was not a good witness under cross-examination in that he confused some of his references. At this distance I cannot reproduce the atmosphere of the trial on which Dr Exner so triumphantly relies. In addition to the Galesburg and Quincy study he makes many references to evidence and cross-examination relating to later studies shortly to be mentioned, and charges of 'evasion', 'misrepresentation', 'warping of facts to suit convenience', 'lack of any sense of responsibility for scientific accuracy or forthright reporting', etc., etc., flow freely from his pen. I have obtained a copy of the judgment which includes a lengthy review of the evidence[7]. It was, incidentally, affirmed on appeal. In this judgment the Master in Chancery characterised, in relation to the case supported by Dr Exner as a witness, 'all of the above contentions and points thus raised by plaintiffs . . . as trifling minutiae which cannot change the obvious and inevitable conclusions presented by defendants' experts:' and in many places he referred to Dr Dean with obvious approval and expressly adopted his evidence.

244. I can only conclude that Dr Dean's credit was not regarded as in any way impaired by the matters to which Dr Exner has referred and in this regard I think the judgment is a better guide than the partial references to a transcript which I have not seen. I have read the analysis of Dr Exner with care. I have read and reread the papers of Dean to which it relates. I am impressed by the obvious care with which, as a pioneer in the epidemiological study of caries, he sets out in meticulous detail the efforts made to eliminate variables. If the acuity of hindsight shows that his efforts were not completely successful, the critics should first ponder the difficulties inherent in such studies. The Galesburg water history was either overlooked or its significance not appreciated, or he was right, it had no significance. But accepting as I do these early studies, not as statistical conclusions arrived at with mathematical precision but as indicating trends, their value is not destroyed by criticism of this nature. In fact it is more destructive of the critic than of the subject criticised.

245. Dean himself regarded this as no more than an initial study. No hasty conclusions were drawn. He himself said:

> 'While these findings gave considerable impetus to the general principle of mass control of dental caries through the public water supply, it was felt that judgment should be withheld until it was determined whether or not the factor, presumably fluoride, inhibiting the initiation of dental caries was operative at a level sufficiently low to eliminate even the milder forms of endemic dental fluorosis'[9].

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Section (c)—The Study of 21 Cities in 4 States

246. This led to the next critical study known as 'The Study of 21 cities in 4 States'. Dean charted the purpose of the study thus:—

> 'The sharp difference in the prevalence of dental caries between the white urban school population of Galesburg and Quincy (Ill.) constituted a striking demonstration of this naturally occurring phenomenon. The inferences from this study interpreted in the light of all the evidence available at that time (1939) pointed presumptively to

> fluoride as the inhibitory agent. The next step therefore, was to expose this natural phenomenon to various kinds of verification. For the purpose of clarifying by detailed studies some of the questions arising from the analysis of the Galesburg-Quincy study and earlier studies, a relatively detailed epidemiological study was planned. Two of the questions upon which information was particularly desired were:—(*a*) To determine whether or not there were domestic waters low enough in fluoride to eliminate the complication of mottled enamel, but still sufficiently high to markedly reduce the incidence of dental caries; and (*b*) to verify this natural phenomenon in respect of other conditions and in a large number of cases over a wide range of circumstances '[1].

This was a large scale project. The results are embodied in two papers[2, 3]. The studies followed the pattern of that of Galesburg and Quincy excepting that only children with continuous exposure to the communal water supply were examined. The same careful consideration was given to the exclusion of variables. The first study gives findings in respect of 2,832 children, residents of eight suburban Chicago communities, the second in respect of 4,425 children in thirteen other cities, Galesburg and Quincy being included. From these findings Dean and his co-workers drew the conclusions expressed in Fig. II, the figure being a correlation between the DMF rates of the children examined with the mean fluoride content of the public water supply in each of the twenty-one cities studied.

247. The data from which this mean fluoride content was calculated is explained in great detail. No attempt is made to gloss over the difficulties in extrapolating data so as to arrive at a figure which ideally should have reflected a monthly mean over fourteen continuous years. Obviously, a continuous water history making possible such an ideal would, generally speaking, be unobtainable but such are the difficulties of epidemiological studies.

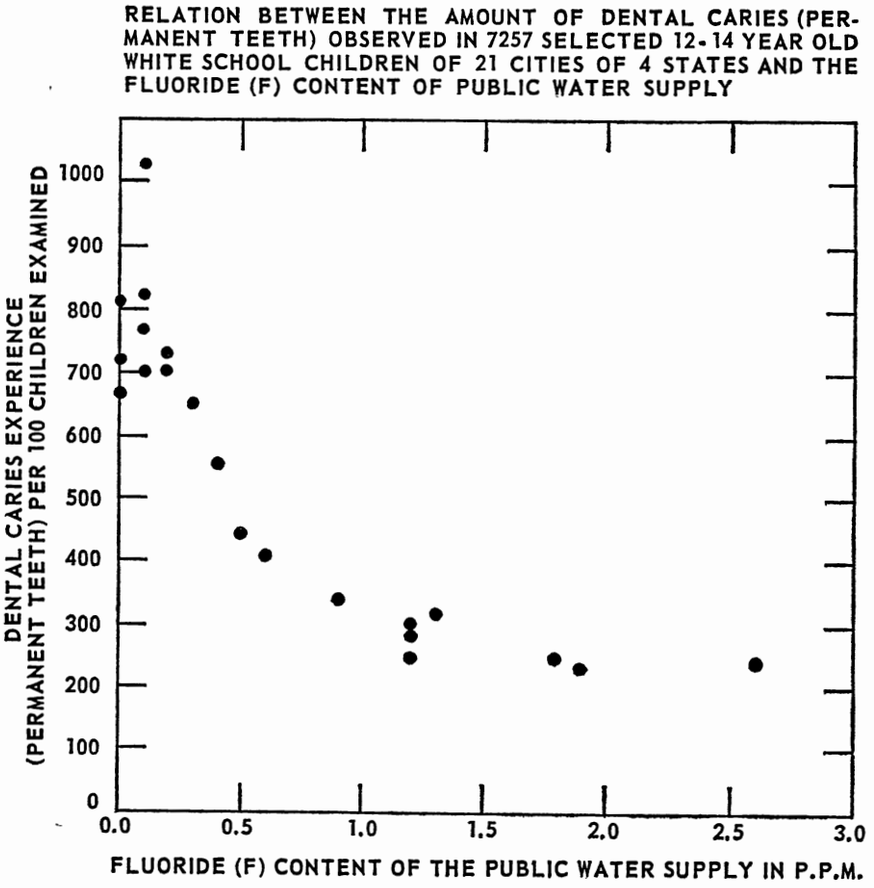
248. These studies and their conclusions have also been criticised. The criticism placed before me, again mainly by Dr Exner[4], relates principally to discontinuous water histories and draws heavily on evidence given by Dean thirteen to twenty years later when cross-examined in the suit already referred to[5], in another suit before a Californian Court (1955) and in an investigation before a Congressional Committee (The Delaney Committee (1952))[6]. I have studied the excerpts of the cross-examinations to which Dr Exner has referred. Again I note the effect was not to displace the confidence in the witness by the judicial tribunals before whom the suits were conducted. Though the aim of it was otherwise, to me the effect of the cross-examination is not so much to throw doubt on the honesty and objectivity of the published reports as to show that Dr Dean was capable of being easily confused in his references to them many years later. He was no doubt a poor witness; the syndrome is not an uncommon one.

249. But, reading his original work, not as he sought to remember it twenty years later, but as it was written at the time, I am of the opinion that the overall effect of such monumental studies as establishing clear indications or correlations of significance is not really destroyed by such flaws in the data used as have been either demonstrated or suggested. In 1957 the Council on Drugs and the Council on Foods and Nutrition of the American Medical Association, at the direction of the House of Delegates of the Association, made an independent review of these water histories in the light of the criticism directed at them by Drs Exner and Waldbott[7]. They reported (and the House of Delegates approved the Report)[8]:

> 'Strong exception has been taken to the water histories given by Dean for several of these cities. We find, however, that they are remarkably helpful even considering the possible looseness of such records. They enable one to make a fair estimate of the average fluoride content of the municipal water supplies four to fourteen years before the examination, the period in which the enamel calcium of the 12, 13 and 14-year-old children was being laid down. Our estimates of the average fluoride content, based on the published statements, were made independently of Dean's and independently of the dental findings; they differ from his only in some minor respects.'.

250. In any case most of the criticisms made, whatever might have been their effect at the time when the studies were made, are now of historical interest only, for my purpose their value must be assessed not only in the light of events at the time but in the light of subsequent events as well. In other words, if subsequent work has confirmed the conclusions drawn by Dean, what's the use of it all except possibly to the historian?

FIG. II.



Taken from Dean *et al.* (1942)[3].

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Section (d)—Other Studies

251. As a result of this study, Dean and others concluded that 1ppm of fluoride represented an optimum level beyond which no advantage was to be gained in caries reduction and which was low enough to reduce both the prevalence and degree of dental fluorosis to limits that would in the public interest be acceptable. Subsequent work (Deatherage (1943)[1] and Nevitt *et al.* (1953)[2]) has confirmed and not destroyed this conclusion except to refine the optimum scale to accord more closely with differences in regional temperatures. These basic conclusions of Dean and his co-workers finally exemplified in the twenty-one cities study that there was an inverse relation-

ship between the prevalence of caries and the amount of fluoride in the water, have been confirmed by many workers in many lands. The literature is so voluminous that in the words of Hodge:—

> 'No exhaustive authoritative summary has ever been prepared of the reports linking fluoridated water to a decrease in the attack rate of human dental caries.'.

In a 'representative' table he lists thirty-five reports relating to drinking water alone. Three, all produced to the Commission, are interesting enough to mention. They relate to Finland[4], Czechoslovakia[5] and to the U.S.S.R.[6]. In the latter the statement appears:

> 'Dean's observation was confirmed in every instance.'.

252. The next step was to test the conclusions drawn.

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CHAPTER 16. FLUORIDATION TRIALS

Section (a)—Four Major American Trials

253. It must not be thought from this summary treatment of the work that preceded the trials about to be mentioned that I have in any sense described the sum total of the epidemioligical or the experimental information available at the time. There already existed a literature of formidable proportions. Roholm[1] had published his monograph on 'Fluorine Intoxication' in 1937. Of this it has been said it divides knowledge on the subject into two periods, the work prior to 1937 and the work since. If reference be made to his work for a survey and bibliography of the early literature and to works such as 'Fluorine and Dental Health[2]' and to 'Fluorine Chemistry[3]' for details of work published between the time Roholm wrote and 1944, it will be apparent that this was indeed a much researched subject not only in the U.S.A. but in other parts of the world as well[4, 5]. Nor must it be thought that epidemiological surveys of the relationship between naturally fluoridated water and caries incidence stopped with Dean's studies of the twenty-one cities. The relationship continued to be verified and established beyond doubt by studies conducted all over the world. As an example only, twelve such surveys for American cities or groups of cities are listed in the monograph 'Fluoride Drinking Waters'[6] as occurring between 1944 and 1959, but such studies were not confined to the United States[7]. It is to be remembered that communities using water naturally fluoridated at significant levels were not exactly unique. A census (1959)[8] of community water sources in the U.S.A. which contained natural fluoride revealed that 895 such communities with a total population of over four million people had been exposed to optimum amounts (0.7-1.1 ppm), the duration of exposure being upwards of seventy-five years[9, 10]. There were also significant populations in the United Kingdom[11], Europe, Canada[12], Asia and South America[13] and elsewhere.

254. Fluoridation, i.e. the deliberate addition of the fluoride ion to communal water supplies as a caries preventative measure, may be said to date from 1945. The decision to proceed with fluoridation trials was made in three United States cities and in one in Canada at approximately the same time. I think it is important to realise that these were individual decisions of the cities concerned. While there was much co-operation and consultation, this was not a centralised experiment under one control, but four separate projects, each one of which was the responsibility of a different authority. The object of all trials was the same, to lessen caries prevalence and severity by adjusting the fluoride content of the water supply to 1 ppm. The statistical demonstration of the results presupposed an elimination of variables and to this end the trials all sought to adopt the technique of a controlled experiment.

255. This necessitated the selection of a control community drinking unfluoridated water (with no other distinguishing chemical characteristics if possible) and conforming as nearly as possible to the racial, social, climatic and economic conditions of each study group, a prefluoridation survey of both groups to establish base data and thereafter periodical and progressive surveys to establish comparative data. Methods of examination and of recording results had to be standardised as far as possible. Reflecting that the period of time over which the trials were to extend was envisaged as extending from fifteen to twenty years[14], it will be appreciated that these were formidable requirements.

256. The four trials to which I refer as the major trials of the period were:—

(i) Grand Rapids, Michigan (control city Muskegon in the same State 0.2 ppm). As another control, comparative data in the opening year of the trials was also obtained from the city of Aurora which had a naturally fluoridated water supply of 1.2 ppm. In 1951, Muskegon decided to fluoridate, so it ceased to be a control in the strict sense. Comparisons were continued however with Muskegon for the next three years and thereafter they were maintained with the base data for Grand Rapids and for Aurora, the final report for the full fifteen years' study being published in 1962[15]. The conclusion was that after fifteen years total caries experience was lowered by fifty to sixty-three per cent in children aged 12-14 years and by forty-eight to fifty per cent in children 15-16 years. The study was criticised by Dr Sutton[16] in 1959 mainly on account of differences in sample sizes, methods of sampling, the adequacy of the controls—particularly after 1951—and in respect of the methods used to assess and eliminate examiner variability.

(ii) Newburgh, New York (control city Kingston). In this case an optimum level of 1.2 ppm of fluoride was selected. The final report after ten years of fluoridation[17] found a 58% reduction in DMF rates for children ages 6-9 who had had continuous exposure and less but substantial reductions for those who had had exposure for part of life only. It has been criticised by Dr Sutton[18] for non-comparability of water supplies, later examination of control city, for variations in methods used and in the examiners and for errors in statistical method.

(iii) Evanston, Illinois (control Oak Park). The report issued after fourteen years of fluoridation[19] i.e. up to 1961, is more detailed than the others. Decreases in DMF rates comparable with those claimed in the other studies mentioned, both as between the base data for the fluoridated group and as between that group and the control group, were reported. Again this study has been criticised by Dr Sutton[20] for failings in the comparability of the control, for alleged discrepancies in reported figures and other methodological errors. Perhaps I should add, in relation to the second matter, as two figures to which Dr Sutton drew attention were rather large, an explanation was sought by Professor Storey of the authors and has been given to the Commission[21]. It satisfies me that, though Dr Sutton quite rightly challenged them, they are not particularly significant.

(iv) Brantford, Canada. No control city was selected before fluoridation at 1 ppm commenced in 1945 (the concentration was raised to 1.2 ppm in 1949[22]) but two prefluoridation surveys on Brantford itself in 1944 and 1945 supplied base data. In 1946 two control cities were selected, Stratford (naturally fluoridated at 1.3 ppm to 1.7 ppm[12]) and Sarnia[23] (almost fluoride free). In effect there have been two trials, the first an intra group study in which the comparison has been made against the base data established by the prefluoridation surveys of 1944-1945 and the second against the controls established in 1946.

257. To deal with Brantford in a little more detail. The final report for the first (intracity) trial was issued in 1956[25] the last recorded year being 1955, i.e. there are two prefluoridation years and ten post fluoridation years taken into account. As a matter of interest this involved twelve annual dental surveys made by the same dentist involving 56,347 school children. I do not reproduce the results but they show comparable reductions to those claimed in the other studies mentioned. The last report for the second (intercity) trial is contained in an official Report[24] issued in 1963 by the Canadian Department of National Health and Welfare and records seventeen and a half years of fluoride experience. Results for the 16-18 age group were as expressed in Tables XIII and XIV.

TABLE XIII

MEAN TOOTH MORTALITY (MISSING AND IRREPARABLE) PERMANENT TEETH PER 100 CHILDREN

Age Group	*Year*	*Mean Tooth Mortality (Missing and Irreparable) Permanent Teeth per 100 Children and Standard Error of Mean*		
		SARNIA (*No fluoride*)	BRANTFORD (*Artificially fluoridated*)	STRATFORD (*Naturally fluoridated*)
16-18	1963	131.33 ± 10.433	37.92 ± 4.274	41.85 ± 5.758

Inter-City Differences in Mean Tooth Mortality (Missing and Irreparable) Permanent Teeth per 100 Children

Age Group	*Year*	SARNIA, BRANTFORD	BRANTFORD, STRATFORD
16-18	1963	—93.41 SS*	3.93 NS*

TABLE XIV

MEAN DMF (DECAYED, MISSING, FILLED) PERMANENT TEETH PER CHILD

Age Group	*Year*	*Mean DMF Permanent Teeth Per Child and Standard Error of Mean*		
		SARNIA	BRANTFORD	STRATFORD
16-18	1963	10.44 ± 0.215	4.74 ± 0.176	4.19 ± 0.212

Inter-City Differences in Mean DMF Permanent Teeth per Child

Age Group	*Year*	SARNIA, BRANTFORD	BRANTFORD, STRATFORD
16-18	1963	—5.70 SS*	—0.55 NS*

*SS = Statistically significant.
*NS = Not statistically significant.

Taken from 1963 Report 'Dental Effects of Water Fluoridation' by Department of National Health and Welfare, Canada.[24]

The report[24] concludes—

> 'The 1963 survey findings of the Brantford Fluoridation Study are consistent with those of our previous surveys of the native child populations of Sarnia, Brantford and Stratford. A continuing reduction in caries experience in 16- and 17-year-old Brantford children seventeen and a half years after the introduction of water fluoridation in Brantford is shown. The Brantford data for these children are presented in relation to similar data obtained from Sarnia (not fluoridated) and from Stratford (naturally fluoridated).
>
> 'The data indicate that there is essentially no difference in caries reducing effect between a mechanically fluoridated water supply and a naturally fluoridated water supply and that this beneficial effect of water fluoridation extends at least until the eighteenth year of life.'[24].

258. These trials too have been criticised by Dr Sutton,[26] the first for the lack of an outside control and for some typographical and mathematical errors and the second because of the late selection of controls (two and a half years after the commencement of fluoridation), because of factors affecting the comparability of controls and for omissions and mis-statements in reported results.

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Section (b)—Criticisms—Dr Sutton

259. The controversy aroused by Dr Sutton's criticisms has passed the bounds of dispassionate debate of matters of scientific interest. Rebutter has been met by rejoinder and there is bitterness reflected on both sides. To attempt to pursue the debate in all its minute detail is a laborious task to undertake: it would be an intolerable burden to the reader. While I have pursued it to the best of my capacity and its echoes were continuous throughout my Inquiry I have concluded that the present relevance of these criticisms is not sufficient to justify more than general comment.

260. I have considerable sympathy for Dr Sutton. He set out to pursue a limited objective (just how limited has not always been appreciated either by his critics or his supporters). His supporters would say he pursued it with scholarly thoroughness; the attitude of his critics would, I think, be correctly expressed as resentment at a display of doctrinaire pedagoguery. I think both sides deserve consideration.

261. The epidemiological conclusions reached by Dean and others as a result of their observations in naturally fluoridated areas prior to the commencement of any of these trials, were not considered by Dr Sutton. The purpose of his investigation was solely to examine the reported conclusions of the five trials referred to. This he did as a critical exercise with thoroughness and he discovered a number of errors and omissions. The same point has been made in a review of the matter by the 'Lancet' in which the writer commented:

> 'Sutton's investigation should be regarded not so much as an attack on fluoridation itself as a criticism of some lax and unconventional statistical procedures.'[1].

I agree with the comment and suggest that in the appraisal of this dispute within a dispute its implications have been widely overlooked.

262. In setting up the five trials as his target, he used expressions which I think did leave him open to counter attack. He said in relation to these trials that upon the results of them 'proposals to fluoridate domestic water are almost entirely based'.[2] This was seized upon by one reviewer, Dr Galagan, who called it a fundamental error and who proceeded:

> 'Actually, the scientific basis on which this public health measure rests was established solidly before any of the abovementioned projects were started. The preventive effect of long-term exposure to water-borne fluoride on caries experience was observed in literally thousands of children residing in many different communities where the water consumed had picked up the element as it coursed over or through the earth's crust. The long series of investigations documenting the relationship are considered to be classic examples of good epidemiological method—so much so that they are used as case studies in teaching the science of epidemiology.'

To this Dr Sutton replied:

> 'Such proposals are based on two different sets of results—those reported from areas where fluorides occur naturally in the water supplies, and those from trials of the mechanical addition of fluorides to waters in which the fluoride content is very low or absent. The former reports are not considered in this investigation, but Dr Galagan says that they were gathered in such a way that they " are considered to be classic examples of good epidemiological method ". In any case, they are of practical importance only if it is known that the results reported will be obtained when commercially available fluorides are mechanically added to water supplies.'[3].

If the reference 'but Dr Galagan says' etc. is an acceptance of what he says, it is significant: if it is sarcastic, it is unworthy. I am uncertain how it was meant to be read.

263. He does not seek to refute directly the reference to Dean's studies as establishing solidly the scientific basis on which this public health measure rests but distinguishes the matter by saying that such studies relate only to naturally fluoridated waters. He then suggests that the extension of the results established by such studies to projects for artificial fluoridation can only be justified under one or other of two conditions, either:

> (*a*) by the use of experimental trials; or
> (*b*) if it can be 'accepted that it can be established, on theoretical grounds, that the results of artificial fluoridation will be identical with those seen in areas where fluorides are found naturally[3].'

264. The second alternative is not discussed by Dr Sutton in his monograph. In the light of the evidence given to me, I can give no weight at all to any implied suggestion that it may not, so that my firm conclusion is that the second of Dr Sutton's alternatives should be regarded as fulfilled. Perhaps I should add to this that Dr Sutton himself has said quite unequivocally that as far as caries is concerned he doesn't think there is any difference in the effects produced by either natural or artificial fluoridation, though as to possible 'toxic' effects he said 'there could well be a difference. I don't exactly know[4]'. Later in evidence he said:[10]

> Q. *Would you say these five trials did not succeed at all in showing the effect of artificially fluoridated water at* 1 *ppm did in fact have an effect on the incidence and gravity of dental caries?*
> A. No, I think they showed some effect but how much effect I am not prepared to state, that is the trouble.
> Q. *Sufficient effect, if no ill effects were shown, to follow from the ingestion of water at* 1*ppm to be a worthwhile public health measure?*
> A. Yes I think so. If there were no ill effects.

The conclusion is, if no ill effects, sufficient benefit to be justified as a public health measure. This attitude was not made clear in his book.

265. However he is quite right in his monograph (and here the dispute at the present day takes on the flavour of an artificial debate, i.e. one in which the argument is pursued for its own sake irrespective of the merits of the issue) in contending that when these trials were set up they were set up as 'experimental trials' in the strict sense of controlled experiments designed to verify an untested and unproven hypothesis. The fact that their character was later stated to be merely that of demonstrations of a proven procedure does indicate a change of ground on which, as a matter of dialectic, a debater would be entitled to comment, but which is only of significance if it affects the merits of the dispute. In the circumstances of our present knowledge I don't think it does.

266. But having thus established his ' Aunt Sallys ' as controlled experiments, he proceeds to set up a specification of such rigidity that it is doubtful whether any similar experiment in this field, involving so many subjects, over so many years, could ever be free from attack. Projects of this nature cannot be controlled with the strictness of a laboratory experiment and I think Dr Sutton with his zest for meticulous accuracy does lay himself open to a charge of doctrinaire absolutism.[5] However, in his criticism he makes many valid points and I have no doubt that the effectiveness of the ' trials ' or ' experiments ', whatever they be called, is to that extent reduced though not destroyed, and when all is taken into account the trends that they establish are still both consistent and compelling.

267. I appreciate that ' trend ' which I have used before is a vague term and in a subject already overburdened with semantic overtones, I have no wish to start another debate equally arid. I can make my meaning clear in the following way. The critics of these fluoridation trials rely on the old truism that nought plus nought still equals nought. The argument has been so expressed so I make no apology for referring to it in such homely terms. Hence having demolished each separate trial to their satisfaction they rely on the sweeping assertion that the overall effect of this considerable body of evidence is nothing. Such a conclusion, which seems to be influenced overmuch by the sophistry of debating technique, presupposes as essential to its validity that the value of every piece of evidence considered is precisely zero and nothing, not an iota, not even a scintilla more. This in the case of the experiments referred to is manifestly incorrect, even absurd. It is true as Dr Sutton has shown, that in many of these trials, because of epidemiological shortcomings, the authors have fallen short of their total objective; their value may on that account be less than the unity sought between theoretical and practical demonstration, i.e. absolute, but to say that the conclusions that they consistently suggest, while less than absolute, are completely and cumulatively worthless is not a judgment but a negation of judgment. It is proofs of this nature, intrinsically and individually less than absolute, but cumulatively of considerable cogency that I refer to as ' trends '.

268. The strength of an evidentiary chain is not necessarily that of its weakest link. As every piece of evidence, more consistent with a given conclusion than any other is added to others with the same tendency, the chances of any other conclusion being correct become progressively more remote. Criticism which is not followed by appraisal is negative only and this is the weakest of so many of the critics of these trials. They make no attempt to appraise the strength of the evidence and this is greater than they will allow. Admitting the imperfections the sum total of the evidence that remains is not only impressive, it is overwhelming.

269. It could well be that as these trials progressed, a growing realisation of the impossibility of maintaining the strict protocol of a controlled experiment led to a relaxation of earlier aims, a lowering of sights as it were, but I feel the most significant comment on this aspect of the dispute is that Dr Sutton's criticism is destructive only. It is a list of errors of varying significance, with little attempt to discriminate and no attempt to appraise the results in fact achieved. If Dr Sutton sets out to maintain that these trials proved nothing, not even trends, he fails; if he didn't, his purely negative attitude has not helped.

270. I own to some uncertainty as to how far Dr Sutton himself would go in these matters. It is sometimes difficult to distinguish between where he ends and his supporters take over. It is true that Dr Sutton said in conclusion, ' It is possible that a case for fluoridation can be solidly based ' and elsewhere he urged an open mind and it is possible that both his friends and his critics have paid insufficient attention to these statements; but if they have done him an initial injustice in ascribing to him the character of an opponent to fluoridation, he has not really discouraged them.

271. I hope I have made it clear that I regard some of Dr Sutton's criticisms as possessing substance, particularly in relation to the methodological details of these trials, though to others I cannot ascribe the same weight. To avoid a charge of generality I cite some examples. At least two studies are criticised because the base rate of the controls was not established before fluoridation began. They were Muskegon (a difference of less than six months) and Oak Park (a difference of one month) and Kingston (five months). I am completely unconvinced that these delays justified the adumbrations of the text. It is true that later in the monograph he says ' This late examination of the control cities on first thought, may not seem to be of much consequence. However, it means that, in all of these studies, a matter of fundamental importance was disregarded—it could not have been established that the children of similar ages in the test and the control cities, prior to the commencement of the experiment, had reasonably comparable caries attack rates '. With respect, in the circumstances of the practical difficulties of such matters this appeals to me as a molehill turned into a doctrinaire mountain. Similarly much is made of the late com-

mencement of the second trial in the case of Brantford. Again undesirable from the point of view of method but I accept the rejoiner of one of his critics that this does not deprive the study of value and its effect could only be to understate the benefits gained by fluoridation. The differences in the water supply of Kingston and Newburgh do not justify the use he makes of them. They are not really considerable either in the scale of such matters or in the light of the available evidence as to the effect of such differences. He is not at his best in his reply to Col. Fuller's criticism[6] of his comment in this regard which is an example of debating technique leaving the substantial point unanswered[7].

272. But basically to pursue the details of this controversy at this time of day is only of historical interest. It is profitless to consider Dr Sutton's criticisms in a vacuum, i.e. divorced from the considerations which arise from other studies and trials which have been reported since. The trend from all over the world has been uniform. Questioned as to why he had taken so little interest in this later work his explanation was that he undertook his initial study at the request of his Dean as a statistical exercise; his monograph was published in 1959; he returned to the subject to answer his critics in 1960, and there his researches ended. As he said in evidence, 'I gave up fluoridation in 1960 when I finished with the monograph, and have been working on other things since, and I really haven't studied the matter at all until this came up in January 1967'. His attitude I express in his own words:

> 'My criticisms of the Newburgh, Grand Rapids, Evanston and the two Brantford trials cannot be affected by reports from other ones, and remain valid.'[8].

This may be so if they are to be judged as a sterile exercise in criticism; it is not so if the merits of the matter are sought objectively to be evaluated.

273. He has favoured me with a very full submission but as I understood him, except in relation to the statistical work embodied in his monograph, none of it is based on personal research or experience, clinical or otherwise, and his statistical work stopped in 1960[9]. The singlemindedness with which he has confined himself to the statistical examination of the five trials mentioned to the exclusion of other aspects of the fluoride controversy is reflected in his failure to seek any clinical experience of fluoridation in practice, a matter on which I have already commented. As to the rest of his submission, he has followed the same pattern as other witnesses on both sides of reporting papers written by and data gathered by others as relevant to their own assessment, whether competent or not, of the matters referred to. This of course was an eclectic process and reflected generally the subjective approach. In this of course he was not alone.

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Section (c)—United Kingdom

274. A number of fluoridation studies from many parts of the world were referred to in evidence and are documented in the literature. They are of course of varying depth and one could make distinctions between them in value. Thus, in the United Kingdom, following the report of an official Mission to North America for the purpose of studying fluoridation, a controlled fluoridation trial was initiated in 1955. There were to be three fluoridated areas (in Wales, Scotland and England) and three controls. The results achieved after five years have been reported[1].

275. Dr Sutton said he regarded them as 'obviously superficial' and he took no notice of them. I do not understand the comment. The procedures adopted, the factors which influenced the selection of the controls and other relevant epidemiological data are summarised in appendices to the Report and they are obviously not superficial. However, as the reported results in 1962 mainly relate to children in much younger age groups than those in the other reports which have been mentioned in evidence and are related to deciduous teeth, their value is at yet incomplete. This is not to say that the results obtained in any way throw doubt on the fluoride hypothesis,

on the contrary, the Research Committee of the United Kingdom Ministry of Health which, in consultation with the Medical Research Council conducted the trials, reported that 'The results for children aged 4 and 5 years, which alone can be compared with American results, appear to be in line with them.'[2].

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Section (d)—Holland, Tiel, Culemborg

276. But undoubtedly the most important of all subsequent fluoride trials is that conducted in the Netherlands by Backer Dirks and his associates, generally referred to as the Tiel-Culemborg Study. This was a deliberate controlled experiment in the fullest sense of the word, designed to test the applicability of American data to the social and dietary conditions of the Netherlands. Fluoridation commenced in the city of Tiel in 1953 (1.1 ppm), while the nearby city of Culemborg served as a control (±0.1 ppm natural fluoride).

277. Great care was given to the planning and execution of the study, particularly in relation to methods of diagnosis[1] and to provide safeguards against variables such as were complained of in relation to the American trials referred to in preceding sections. An attempt was even made to grade carious lesions by the depth of their penetration in the direction of the pulp so as to provide an index of severity as well as prevalence. Its methodology received the approval of Dr Polya than whom perhaps there was no stricter advocate of the conditions which should obtain in a scientific experiment[2, 3] and in this instance I am happy to adopt his comment.

278. There have been two reports[4, 5] representing six-and-a-half and eight-and-a-half years of fluoridation respectively. They have been supplemented by information obtained from the Netherlands Ministry giving results achieved after ten years[6]. Though it is not completely clear, I judge that the experiment is now concluded, the view of the senior author being that in view of the information available from other parts of the world (he refers to the American studies and trials mentioned above which he says have been fully substantiated in other countries with a different diet) it would not be necessary, in what may be called an adaptability study, 'to carry on such a study for fifteen years; as soon as the initial results—after four to seven years—are available and shown to be comparable with the American results, every reasonable doubt as to the effect of water fluoridation can be put aside. This means also that the results of studies which have been going on for a longer period of time can be accepted as the ultimate effect of water fluoridation'[5].

279. In the view of the authors, expressed after the first six-and-a-half years period, and allowing for the differences in the methods of examination and in the definition of the caries criteria, the conclusion to be drawn was 'that water fluoridation in the Netherlands has a favourable caries inhibiting effect which is fairly comparable to the results of the American studies after the same period'[4]. There is nothing to qualify and everything to confirm those conclusions in the later reports[5, 6, 7]. As an example, the latest information (i.e., after ten years) claims a percentage reduction as high as 78% for interproximal caries for 12-year-old children of the fluoridated city[6].

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Section (e)—Other Trials and Studies

280. The universally conceded importance of these meticulous Dutch trials makes it really unnecessary to range more widely. There is nothing in the literature that achieves the same degree of accuracy. Nevertheless other controlled studies have been mounted with comparable results and there have been a number of retrospective intragroup studies in which the comparison has been made against baseline data established in respect of the fluoridated population before fluoridation started. Examples are New Zealand[1, 2, 3, 4], Rush City, Minnesota[5], Montgomery and Prince Georges Counties, Maryland[6], and Georgia, U.S.A.[10]. Others are referred to by the British Mission of 1952[9]. A similar study has been initiated in Australia for Canberra which commenced fluoridation in September 1964. However this is only a partial list.

281. In Japan a controlled study commenced in 1952 has been reported by Gen Minoguchi[7, 8], involving Yamashina district in Kyoto and Shugakuin, another district of the same city. Both districts drew from a common water supply, the naturally occurring fluoride content of which was 0.05 to 0.09 ppm. The supply of Yamashina was raised to 0.6 ppm, a level which was fixed after taking the local climatic conditions into account. This is a lower level of fluoridation than would have been indicated by practice in Western countries but as dietetic differences were thought to increase a local susceptibility to dental fluorosis, this lower level was adopted as a conservative step. Results after eleven years are considered to show a result almost equal to that of Grand Rapids at 1 ppm but a little inferior to the results of Newburgh in respect of children over ten years of age[8].

282. In Sweden another controlled trial was conducted from 1952 to 1959. The water supply of about half the population of Norrköping (total population 90,000) was fluoridated. Results after seven years have been reported and confirm the fluoride hypothesis[11]. This trial is the subject of a separate chapter (Ch. 45, *post*).

283. I have not thought it necessary to refer separately to studies carried out in connection with artificial fluoridation at Yass in N.S.W. and Beaconsfield in Tasmania. No control and no base data was established for either place prior to fluoridation and they do not therefore really rank in a chapter which is primarily concerned with the verification by controlled studies of the fluoride hypothesis. I have, however, attended at clinical examinations of school children in both places, as well as in other unfluoridated areas to give myself some point of reference. The results as seen and as recorded are entirely consistent with what is claimed in the more formal studies which have been mentioned.

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Section (f)—Assessment of Benefit

284. I deliberately make no attempt to quantify in exact mathematical terms the benefit in caries reduction that fluoridation may be expected to bring. I have quoted some figures to indicate the order of the benefit claimed in the trials to which I have referred. I would deprecate

attempts to apply them too literally. Fluoridation will benefit teeth and surfaces of teeth selectively and not equally. Its effect will be quantitatively different at different ages and except with the most refined of recording techniques the indices normally in use will fail to reflect severity as distinct from prevalence. That the dental benefit is impressive and substantial cannot be doubted, but in the last resort efforts to measure it are approximations only, some being better than others.

285. Arnold[1] in 1943 forecast that fluoridation would bring about—

(*a*) a fall of nearly 60% in caries intensity;
(*b*) a sixfold increase in the number of children still caries free at school leaving age;
(*c*) a decrease of about 75% in the losses of first permanent molars during this period of life; and
(*d*) a fall of about 95% in caries attacks on the approximal surfaces of the upper incisors.

286. These are impressive claims and much attempt has been made to show that they are overstated. Adler[2] reviewed them in 1967 for the World Health Authority by reference to the published results from the major American trials but with a glance at the Dutch and Japanese studies as well. As he points out in relation to the American trials[2]:—

> 'The results obtained in the four studies are not only basically similar but to a large extent even quantitatively identical. Nevertheless there are differences in presentation, in the time when the interim researches were made in the grouping of the examinees by age, in the findings deemed worthy of communication, etc., etc. Because of this it is not possible to summarize the results in a uniform manner . . . .'.

His conclusions are as follows:—

(*a*) The fall in caries intensity among 12- to 14-year-old children was well up to expectations.
(*b*) As to total freedom from caries, the predicted sixfold increase was substantially exceeded in the two studies to which he refers.
(*c*) As to first molars, most reports contained no comparable data figures. However there were numerous comparable data as to the overall loss of teeth and his conclusion is that in this respect too fluoridation fulfilled expectations, although there are some extraneous factors such as improved standards of dental treatment which may be reflected in the overall improvement.
(*d*) The upper incisor approximal surfaces are not specifically reported on in the four American trials. He draws attention to the fact that in the Tiel-Culemborg study, on a slightly wider basis, the results were again close to those predicted.

287. He does not refer to Dr Sutton's criticisms, nevertheless the consistency with which the first two benefits claimed have been approximated not only in those trials, but in trials and clinical studies of all types in all parts of the world makes them impossible to gainsay. In a later chapter (Ch. 43) of my report I take into account specific arguments that the caries lessening effect is a matter of a few years delay at most and that any benefits gained in childhood are not retained in adult life. Anticipating that discussion, I conclude that similar benefits to dental health of the same order as are mentioned in the preceding paragraph are certain to ensue from fluoridation to an optimum level in this State.

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Section (g)—World Status of Fluoridation

288. Appendix 'H' prepared by officers of the Commission is an attempt to present in summary form the present status of fluoridation in the countries of the world. It is probably not complete but so far as it goes it is I believe accurate. Considerable efforts have been made by reference to Embassies, Health and other Government authorities to get the most up-to-date information available. The American data which is the most comprehensive is as it appears in the United States Public Health Service Fluoridation Census for 1966[1]. Other references are given in the Appendix.

TABLE XV

FLUORIDATION IN THE UNITED STATES 1945–1966

INSTITUTION, DISCONTINUANCE, AND REINSTITUTION OF CONTROLLED FLUORIDATION

Year†	Fluoridation Status at End of Each Year			Fluoridation Instituted Whether or Not Discontinued			Fluoridation Discontinued Whether or Not Reinstituted			Fluoridation Reinstituted After Discontinuance		
	Number of Communities	Number of Water Supply Systems	Population*	Number of Communities	Number of Water Supply Systems	Population*	Number of Communities	Number of Water Supply Systems	Population*	Number of Communities	Number of Water Supply Systems	Population*
1945	6	3	231,920	6	3	231,920	..	..	..	..	..	..
1946	12	8	332,467	12	8	332,467	..	..	..	..	..	..
1947	16	11	458,748	16	11	458,748	..	..	..	..	..	..
1948	26	13	581,683	26	13	581,683	..	..	..	..	..	..
1949	49	29	1,062,779	49	29	1,062,779	..	..	..	..	..	..
1950	100	62	1,578,578	101	63	1,595,128	1	1	16,550	..	..	..
1951	368	171	5,079,321	370	173	5,108,771	2	2	29,450	..	..	..
1952	751	353	13,875,005	758	360	14,079,130	7	7	204,125	..	..	..
1953	1,007	482	17,666,339	1,019	494	17,751,207	14	14	256,399	2	2	171,531
1954	1,194	572	22,336,884	1,226	602	23,528,254	36	34	1,367,021	4	4	175,651
1955	1,347	672	26,278,820	1,407	719	27,883,734	66	53	1,795,464	6	6	190,550
1956	1,583	772	33,905,474	1,656	828	35,672,794	83	66	1,999,575	10	10	232,255
1957	1,717	879	36,215,208	1,801	938	38,124,663	97	72	2,215,118	13	13	305,663
1958	1,890	995	38,461,589	1,986	1,060	40,463,466	111	80	2,311,813	15	15	309,936
1959	1,990	1,081	39,628,377	2,093	1,153	41,801,740	120	89	2,509,084	17	17	335,721
1960	2,111	1,172	41,179,694	2,221	1,251	43,390,924	127	96	2,546,951	17	17	335,721
1961	2,197	1,249	42,201,115	2,301	1,328	44,418,750	130	99	2,563,530	26	20	345,895
1962	2,321	1,350	44,045,392	2,429	1,433	46,289,156	134	103	2,589,659	26	20	345,895
1963	2,612	1,482	46,678,380	2,723	1,567	49,002,866	143	111	2,760,035	32	26	435,549
1964	2,758	1,573	48,363,066	2,869	1,659	51,179,019	152	120	3,466,178	41	34	650,225
1965	3,030	1,692	59,855,024	3,141	1,786	63,873,219	163	127	4,905,568	52	38	887,373
1966	3,145	1,785	62,427,290	3,257	1,872	66,446,000	165	129	4,961,594	53	42	942,884

*Includes Adjustment for Population Growth Based on the Bureau of the Census Population Estimates.

†Year reported to USPHS.

Taken from United States Public Health Service, Fluoridation Census for 1966.[1]

289. Counting heads may not be a very good argument, but in assessing the probability of harmful effects remaining undetected it is at least of interest to know that in the United States alone some 62½ millions are currently using artificially fluoridated water while another 10 million are using water naturally fluoridated at 0.7 ppm or higher.

290. Opponents of fluoridation place emphasis on the number of communities, principally in the United States of America, which having once fluoridated have at some later stage reverted to a non-fluoridated water supply. The incremental position in that country is as shown in Table XV.

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Section (h)—Conclusion

291. To sum up this chapter of my Report. The epidemiological work which culminated in the 21 City Study by Dean *et al.*[1] in 1942 has been confirmed and is so widely accepted that it cannot seriously be doubted[2, 3, 4]. It established an inverse relationship between caries prevalence and the presence of fluoride in the water and suggested that the optimum protection with the least degree of dental fluorosis was achieved at 1 ppm. Subsequent work, e.g., Galagan[5] and Minoguchi[6] has shown that this figure may be capable of some modification to accord more closely with climatic or dietary differences.

292. Dean's observations related to communities where fluoride occurred naturally in the water supply. There is no evidence to suggest that such fluoride when in solution differs in any material way either chemically or physiologically from fluoride deliberately added to the water. Though this is expressed negatively, the evidence is affirmative on this point that there is no such difference, and, for reasons expressed in a later chapter, I reach this conclusion despite any alleged association between fluoride and 'hard' water.

293. The four experimental fluoridation trials of Grand Rapids, Newburgh, Brantford and Evanston launched in 1945 were designed as controlled trials to test the fluoride hypothesis established by Dean. Originally intended to be pursued for ten to fifteen years, after five years experience only, fluoridation was adopted officially by the U.S. Public Health Service in 1950[7]. The story how this came about is told by McNeil in 'The Fight for Fluoridation'[8]. Approval by the American Dental Association followed in November of the same year. Both bodies had hitherto urged restraint until the results of the experimental trials were known. Dean was at the time Chief of the National Institute of Dental Research and according to McNeil had been the chief influence in restraining the proponents who had wanted the decision made before.

294. That both these bodies acted so quickly after five years' results instead of waiting for the full fifteen is a matter of history. It seems fairly clear that they did so because the five years' results satisfied them that they were justified in so doing[9]. As a matter of abstract epidemiological method they may have been wrong to have acted so precipitately but as a matter of practical public health nothing has occurred which casts any doubt on the correctness of their decision. In other words the most that can be said is that they anticipated results; it cannot be said from the point of view of merits of the matter that they reached a wrong decision. Statistically however the studies on which they relied possessed imperfections and flaws, and they have been criticised by Dr Sutton. His criticisms, however, are negative only. While lessening the value of these trials as proofs of the hypothesis they set out to test they do not establish any counter proposition.

295. Viewed not as controlled experiments but as demonstrations of the value of fluoridation, these tests manifest a uniform trend of caries prevention and reduction consistent with Dean's observations and Arnold's predictions. The same trend has been confirmed extensively, indeed subsequently I know of no satisfactory evidence suggesting a contrary conclusion. One experiment in Holland, designed in respect of duration as an 'adaptability' test and not as a fundamental verification, though full results for seven-and-a-half years only are given, supplies meticulously the formal methodological accuracy that the earlier trials lacked. It confirms fully the trends shown by them.

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CHAPTER 17. THE LEVEL AT WHICH WATER SHOULD BE FLUORIDATED

Section (a)—General Considerations

296. It is accepted that the most sensitive indication of an excessive intake of dietary fluoride is dental fluorosis or, as it is sometimes but unfortunately called, mottling. I say unfortunately because, in the milder forms of the condition, the term conveys an inaccurate picture. Hence the optimum intake may be defined as that which will afford to a given population the greatest protection against caries with the least degree of dental fluorosis. To determine the level at which dental fluorosis may become an objectionable feature is therefore a prerequisite of any fluoridation proposal. This is a subject of such importance that it will be dealt with separately (Part 'G', Ch. 21, *post*).

297. It is important to keep in mind that the disturbance of calcification which leads to mottling of permanent teeth (it seldom affects the deciduous dentition) occurs in the pre-eruptive stage of tooth formation during calcification of the crown[1]. If one excepts the third molars or wisdom teeth this period may be taken to be from infancy to approximately the eighth or ninth year[2]. The drinking of fluoridated water at any later stage will not lead or contribute to dental fluorosis. This is sometimes overlooked or insufficiently appreciated as appears from a letter appearing in the Australian Medical Journal as recently as 11 May 1968[3]. Because of this age factor, arguments which postulate an excessive consumption of fluoridated water such as may be due to addiction to beer or tea, are not generally relevant in the context of mottling. The threshold of fluoride intake beyond which skeletal fluorosis may be expected in adults is much higher as will be seen by reference to Chapter 22 (*post*).

298. The amount of fluoride ingested by an individual will depend on the amount absorbed from three main sources—water, food and the atmosphere. The last is not significant except in areas of gross industrial pollution. Food, though containing fluoride, is not an effective dietary source in that most is bound in insoluble form such as apatite. However some will be absorbed and an allowance has to be made for the amount taken up in this fashion, and in some parts of the world, e.g., Japan, where there is a possibility that dietary habits may exert a significant effect, e.g., by a large consumption of fish[4], this may have to be allowed for as special factor. As far as Australian diet is concerned in general and Tasmanian in particular, I have seen no reason on the evidence to doubt the relevance of the information available in respect of the U.S.A. and Canada, the United Kingdom and New Zealand, as reflecting generally similar dietary habits and standards. A similar conclusion is implicit in the recommendations of the National Health and Medical Research Council of Australia in relation to the matter[5]. There are also reports from which may be assumed a relationship in some areas of the world, particularly in tropical and subtropical zones, between a heightened susceptibility to dental fluorosis and chronic malnutrition. Such areas have been found in India, Arabia and in post-war Italy. They will be mentioned again (Ch. 22 (c), *post*) but for the moment I will say that I do not think they have any immediate relevance to fluoridation at 1 ppm in a Tasmanian community.

299. Drinking water remains the most significant source of dietary fluoride. At levels below 8 ppm almost all fluoride in solution is readily absorbed by the gut, even in 'hard' waters. The amount of water drunk is therefore the primary determinant of fluoride intake and this will be affected by climatic factors, in particular by temperature which may affect the body's demand for fluids. The manner in which this is taken into account in determining the optimum level of fluoridation for any given community is discussed in the next section.

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Section (b)—Temperature as a Determinant of the Fluoridation Level

300. Dean's original work in the estimation of an optimum level of water-borne fluoride at about 1 ppm[1] was based on ten Mid-Western Counties in the U.S.A. with a mean annual temperature of about 50° F. It was recognised that higher temperatures might require a lowering of the optimum[2, 3, 4] and adjustments to this end had been applied in fluoridation practice, though the means by which such adjustments were determined, varied.

301. The definitive studies are those of Galagan and his associates. In the first study[5] using data from the '21 Cities Study' by Dean[6] he compared the DMF rates and the amount of dental fluorosis as reported by Dean with that found by his investigators using similar methods in children of the same age groups in six Arizona communities having fluoride concentrations at about the same level as Dean's cities but a much hotter climate (70° mean annual temperature as against 50° in the cities studied by Dean). The results indicated that the children born and raised in the higher temperature zones consumed approximately twice as much water as those in the lower. He concluded that in the context of fluoridation, temperature was the most important element of climatic environment and that fluoridation levels should be adjusted so as to reflect differences.

302. Further studies[7] followed in which fluid intakes of children were studied in relation to temperature and as a result the authors formulated an equation which expressed the relationship between mean maximum temperatures and water intake. In a final study Galagan & Vermillion (1957)[8] combined the results of these studies to produce a formula from which the optimum level of fluoride for a particular area could be calculated according to the mean annual maximum temperature.

303. In practice the results are expressed in Table XVI which has been enlarged to include lower and upper control limits as approved by the U.S. Public Health Service. It is not however an exact reproduction of the formula as temperature ranges are given within which optimum values may be found which do not follow the exact values obtained by the use of the formula. But if taken to the nearest tenth of a millionth part they agree.

TABLE XVI*

RECOMMENDED FLUORIDE CONCENTRATIONS IN DRINKING WATER FOR APPROPRIATE CLIMATIC CONDITIONS AFTER GALAGAN & VERMILLION (1957)

Annual average of maximum daily air temperatures†	Recommended control limits Fluoride concentrations in Mg/L (ppm)		
	Lower	*Optimum*	*Upper*
50.0–53.7	0.9	1.2	1.7
53.8–58.3	0.8	1.1	1.5
58.4–63.8	0.8	1.0	1.3
63.9–70.6	0.7	0.9	1.2
70.7–79.2	0.7	0.8	1.0
79.3–90.5	0.6	0.7	0.8

* United States Public Health Service. Drinking Water Standards Revised, 1962.

† Based on temperature data obtained for a minimum of five years.

They are the levels recommended by the South African Commission[9].

304. Mean maximum temperature is the average of the highest reading in each 24-hour period throughout the month. For the year it is the average of the monthly readings so obtained[10]. This figure is used preferentially because it relates to day-time temperatures which have more effect on water consumption[11, 12]. For Hobart for the thirty-year period 1931-60 it is 61.8° F; for Launceston for a twenty-six-year period, 64.8° F. Mean temperature uses the daily average temperature, i.e., both minimum and maximum instead of the maximum only. For Hobart the yearly figure is 53.9, Launceston 54.8. Mean temperature as an index was used in the earlier study[5] of Galagan's that has been referred to. Confusion between the two is possible unless the distinction is kept in mind. Entering Table XVI with the mean maximum figures for Hobart and Launceston, optimum levels are 1 ppm and .9 ppm respectively, with an acceptable range according to the U.S. Public Health Service of 0.8 to 1.3 and 0.7 to 1.2 ppm respectively. By formula the comparable figures are .985 and .934 ppm.

305. In 1967 Richards *et al.*[11] reported a comprehensive five-year study in California. The survey involved more than 9,000 children between 12 and 14 years of age, distributed between an eighteen-zone grid of three mean maximum temperature ranges, —65° F or lower, 66-79° F and 80° F or higher, with six fluoride levels ranging from a minimum 0.15 ppm to 1.8 ppm maximum. Table XVII embodies their findings, these being in the author's view 'the fluoride-temperature ranges that will provide substantial protection against dental decay with the minimum possibility of producing objectionable fluorosis':—

TABLE XVII

RECOMMENDED ANNUAL AVERAGE FLUORIDE ION CONCENTRATION FOR DOMESTIC WATER SUPPLY

Temperature (Mean maximum)	Optimum fluoride concentration range (ppm)		
	Minimum	Optimum	Maximum
65° F or lower	1.1	1.2	1.3
66-79° F	0.8	0.9	1.0
80° F or higher	0.5	0.6	0.7

From Richards and Others[11].

The use of this table would indicate an optimum of 1.2 for both Hobart and Launceston.

306. The matter of optimum levels had been reviewed in 1954 by the National Health and Medical Research Council of Australia[12]. Provisionally, and using a different temperature index of 'mean normal maximum temperature' calculated by reference to maximum mid-summer and mid-winter temperatures, they had recommended an optimum figure for Hobart of 1.2 ppm which was verified by reference to Galagan's 1953 paper; but this was before the definitive work of 1957 had appeared. Nevertheless it agrees with the levels suggested by Richards *et al.*

307. Yet another writer, Gen. Minoguchi[13, 14] has recently expressed the view that Galagan's formula, which is incidentally the lowest, may be too high. He has suggested a mathematical recalculation based on mean annual temperatures to give slightly lower results which accord with his fluorosis findings in relation to the Japanese city of Kyoto. He does not take into account the work of Richards *et al.* and in any case the reductions suggested are very slight.

308. The findings of Minoguchi have not been commented on by the witnesses and in the form in which they appear in the projected World Health publication they came to hand too late to enable this to be done. As Minoguchi himself acknowledges, the Japanese evidence may be related to dietary and nutritional features not applicable to American or Australian diets. Until this Japanese study has been evaluated I am uncertain as to the significance that should be attributed to it. It is a factor however that contributes to my reluctance to recommend in relation to Tasmanian fluoride levels any alteration in an upward direction as some have suggested might be necessary.

309. One argument that constantly recurs is based on the likelihood of individual variation in fluid intake. Between adults it may be large, but so is the safety margin between normal intake and a toxic level (see Ch. 26 (*post*)). Between children it may still be significant[15, 16, 17] and as in children the safety margin in relation to dental fluorosis—the most sensitive index of excessive intake—is much smaller, being generally estimated as twofold, it is of more importance.

310. At this stage it is necessary to anticipate. There are two controlling factors implicit in the determination of an optimum level of fluoride. The first is the cariostatic effect, the second is the degree of dental fluorosis that may result. The aim is to achieve the maximum of one compatible with a minimum of the other under given climatic conditions and the resultant product is the optimum level. As such it is a compromise between a high (not a maximum) degree of caries prevention with a tolerable level of dental fluorosis. A predictable relationship between these two factors and fluoride at different levels has been demonstrated in a number of community studies in different temperature zones. As community studies they in themselves take into account a wide range of individual variation in dietary habit but as community or mean figures they cannot precisely match the individual need of all. That is, it must be acknowledged that some individuals will eat and drink more than others, some will receive more fluoride than the optimum, some less. This is inescapable. In those children who receive more, if their total intake of fluoride is more than the optimum for their age, dental fluorosis may occur[17]. Fluorosis even in the less noticeable forms, is a sensitive index and occurs at lower levels of intake than any other toxic symptom. It is by reference to this premonitory symptom that optimum community levels have been determined.

311. Those who receive less, such as children who do not like drinking water or who obtain the great bulk of their fluid requirement from whole milk—the low fluoride level of which is not generally speaking affected by the fluoridation of communal water supplies—will gain some but not the optimum protective effect[18].

312. The question for judgment is, whether the amount of fluorosis that can be expected (less than 10% of 'very mild' (see Ch. 21, *post*)) is in any real sense a detriment or too big a price to pay for the cariostatic benefit that fluoridation may be expected to confer on the child population as a whole. On this subject opponents and proponents differ and will continue to do so reflecting a fundamental diversity of opinion which it is impossible to reconcile. My own considered view, formed after taking into account the views of pediatricians, health advisors and dentists, is that the advantages so far outweigh the slight disadvantage suffered by a very small section of the population that the two matters cannot seriously be compared. Included in the advantages to be gained is a substantial reduction in mottling and enamel defects from idiopathic causes other than fluoride which experience has shown follows from fluoridation and which from any aesthetic viewpoint alone would tilt the scale in favour of fluoride quite apart from any question of caries benefit (see Ch. 21 (a), *post*).

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Section (c)—Whether Tasmanian Levels Should be Altered

313. This has been suggested. It could be the later work of Richards *et al.*[1] indicates a need to raise them and it is faintly supported by some clinical evidence which suggests that in Beaconsfield results may have fallen below the full effect anticipated. Some reliance has been placed on the recommendation (in 1954) of the National Health and Medical Research Council of Australia[2] that the optimum figure for Hobart should be 1.2 ppm but this was a provisional recommendation only and would no doubt fall to be reconsidered in the light of Galagan's (1959) paper. I have a suspicion that there has been some confusion between different temperature indices, e.g., there are three different indices used in Galagan (1953), in the recommendation of the National Health and Medical Research Council (1954) and in Galagan (1957) and the distinctions are sometimes missed[3]. The Japanese formula which would lower slightly the levels obtained from Galagan's 1957 formula uses mean annual temperature however which Galagan used in 1953, but discarded in 1957 as have all others referred to, in favour of mean maximum, as more likely to reflect differences in fluid intake.

314. There is evidence that a drop of as little as 0.2 ppm below the optimum will markedly prevent full dental benefit being obtained[4]. In the study referred to the differential in benefit for 6-year-old children between optimum fluoridation and fluoridation 0.2 ppm below the optimum was as great as 65% to 36%. So that, if a general decision is made that fluoridation be introduced, the accurate determination of the optimum level—and its maintenance—are matters of importance. Anxiety on this score has prompted the question whether in the light of the foregoing circumstances the Tasmanian levels should be revised.

315. I have come to the conclusion no sufficient case has been made out to warrant any recommendation that present levels should be changed. I would suggest that Galagan's 1957 recommendations, which have been widely used, are still the most authoritative guide. Moreover I must point out that performance to date in fluoridation has in Hobart in particular fallen short of intention. The causes are discussed later, but Fig. III shows the level in fact achieved in Hobart over the last three years is no more than an average of .895 ppm. For two years it was not more than .87 ppm. However these figures relate to fluoride introduced. Levels at outlet would possibly be .05 less[5]. It is clear that the intended level of 1 ppm which at outlet is the indicated level for Hobart has not with regularity been maintained and that the overall reduction below the optimum level, determined according to temperature range though slight is substantial enough to warrant some pessimism as to the likelihood of maximum dental benefit being achieved. On the other hand the difference between optimum levels and those levels at which an unacceptable level of dental fluorosis may result is possibly no more than twofold[6], so that any increase in an upward direction is a matter to be approached cautiously. I feel it is better to see what can be achieved at 1 ppm regularly maintained at outlet than to speculate on higher levels at the moment.

316. It is to be noted that the appropriate level of fluoridation for Northern Tasmania according to Galagan's formula is .94. Performance in Beaconsfield to date has been slightly but not significantly below as in the case of Hobart. In these circumstances no special comment is necessary. I think .9 ppm should be substituted for the present target level of 1 ppm until it can be clearly seen in the light of experience whether any further adjustment is necessary.

317. The question of a seasonal adjustment to fluoride levels is one that has been raised from time to time in the interests of a more precise adjustment to temperature-caused variations in fluid intake. In Western Australia temperature variations between various zones in the State are in fact being allowed for by variations in fluoride concentrations, not only as between different areas of the State but as between winter and summer[7]. The summer levels reflect mean temperatures which are hardly applicable to Tasmania. While the matter should be kept under review, I do not think at the moment that the evidence warrants for Tasmania any such refinement as a variation between summer and winter levels[2] particularly as the prevailing climatic conditions are not extreme.

318. While I make no recommendation in the matter of raising present levels I do not regard them as immutable. They are a proper subject for study by the Fluoride Committee which I recommend should be established (if the decision be made that fluoridation be generally adopted as a measure of public health) in the light of future developments. They would be greatly assisted if proper surveys could be mounted to determine the prevalence of mottling, both fluorotic

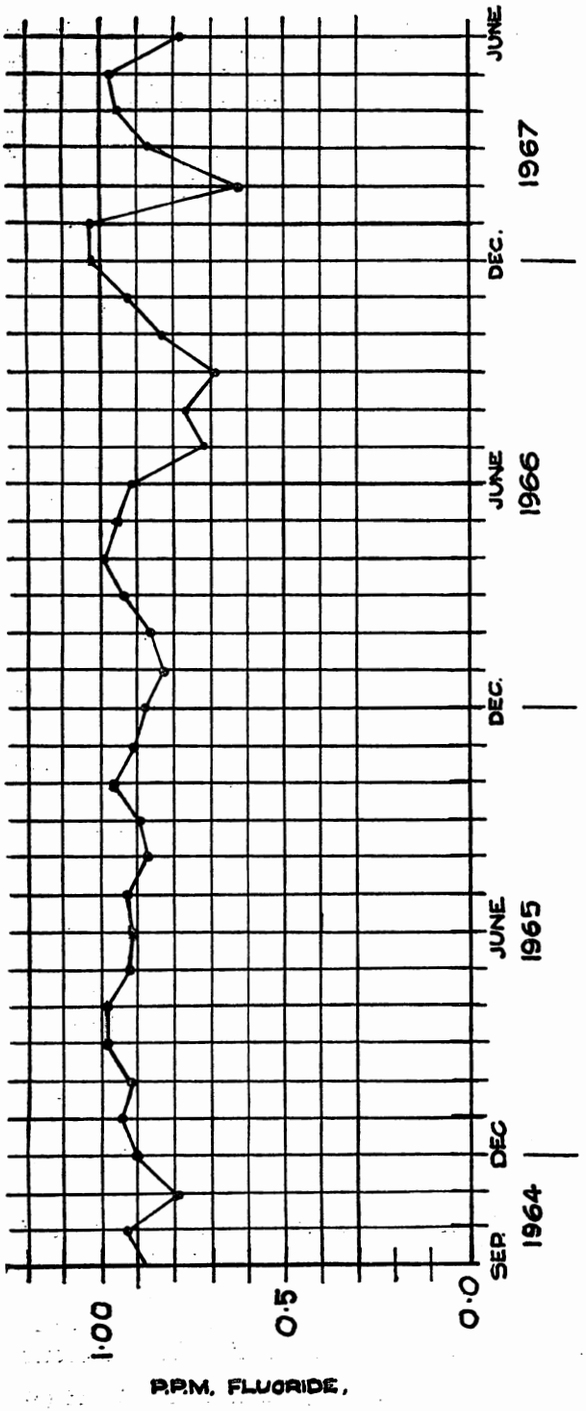
FIG. III

THE MONTHLY LEVEL OF FLUORIDE ADDED TO THE WATER SUPPLY AT HOBART AND WEST TAMAR FROM SEPTEMBER 1964 TO JUNE 1967.

HOBART

AVERAGE LEVELS IN P.P.M. FLUORIDE.

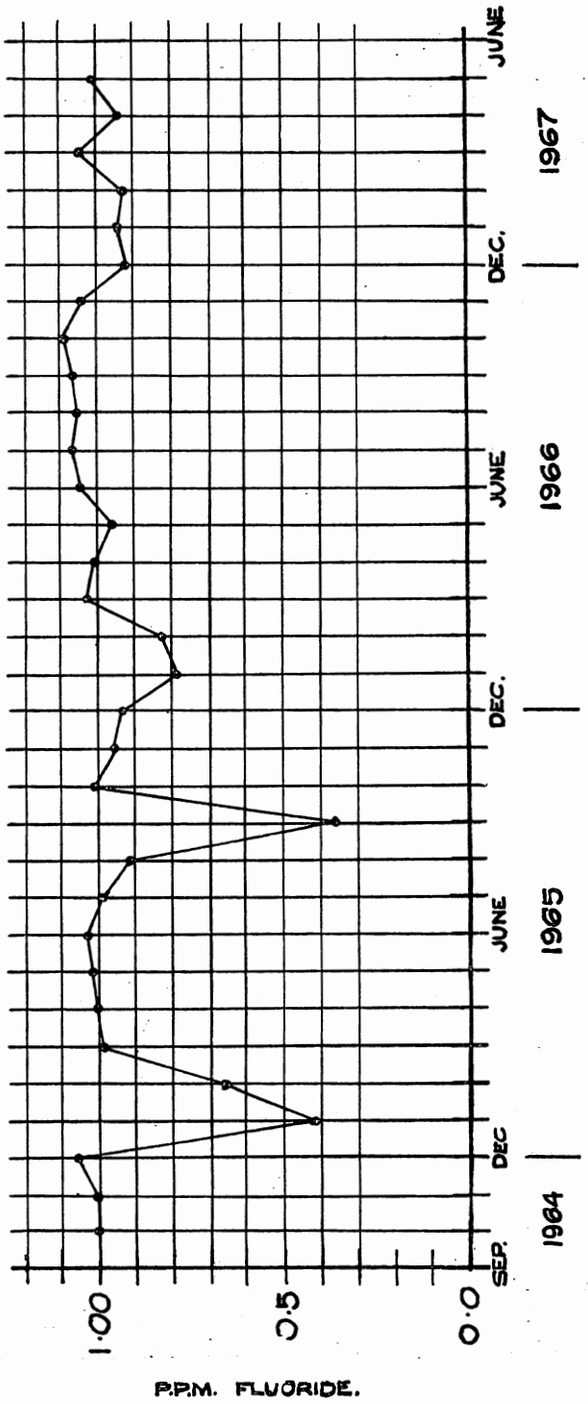
1964 (PART)	·87
1965	·91
1966	·89
1967 (PART)	·87
1964-67 (OVERALL)	·895



WEST TAMAR

AVERAGE LEVELS IN P.P.M. FLUORIDE.

1964 (PART)	1·02
1965	·81
1966	·95
1967 (PART)	·97
1964-67 (OVERALL)	·915



and idiopathic, in selected areas. There is no reason why this could not be done cheaply in connection with present surveys being conducted by the School Dental Service. It is of course essential to the validity of any survey that the pre-determined level of fluoridation should be accurately maintained.

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PART G—TOXICITY OF FLUORIDES

CHAPTER 18. GENERAL CONSIDERATIONS

319. A substance may be said to be poisonous or toxic when on being absorbed by or introduced into the human organism it destroys life or injures health. If, irrespective of dosage, it be correct so to label all substances which have that potentiality, then there is practically nothing in the human environment that might not be considered poisonous in the sense that a level of dosage must ultimately be found that will cause harm. If, on the other hand, the epithet be applied only to that dose or concentration which will bring harmful results, then perhaps there is no 'poison' popularly so called that is not capable of being ingested without harm and even with benefit. Somewhere in the abundant literature on this matter someone has quoted Paracelsus. 'Everything is poison and everything is medicine' and this, I take it, is what was meant.

320. In one usage the term is absolute. No matter in what form or in what concentration, some would have it that fluoride and its compounds are poisons and remain so immutably and irreversibly. Such a usage is popular. It tends to be emotional and because it infers consequences which may not in fact even be possible, it is imprecise. It was exemplified in the following submission from an academic source:[1]

> 'I submit that the argument that fluoride is "not a poison at 1 ppm" (or at any other concentration) with the implication that the word "poison" is correctly used only with reference to a toxic dosage, so that every substance may be a poison at some dosage and beneficial at a lower one, is merely a tendentious misuse of language. The noun "poison" refers to the nature, not the quantity, of a substance. Water is not a poison, though it is possible to poison oneself with an excess; fluoride, arsenic, strychnine, are all well-known poisons because they are harmful, or lethal, at low dosages.'

I do not wish to chop logic, but to say that it is possible to 'poison' oneself with something that is not a 'poison' is not a happy exposition of one's argument.

321. The other use of the term is relative. A substance is poisonous when in relation to a given set of circumstances, concerned principally with chemical form, dosage, the subject to which administered and the manner of administration, it is capable of having the harmful effect that the epithet implies. I would have thought that, in any form of inquiry attempting precision, the relative use would be the only permissible one, but strangely I have a number of submissions founded on the absolute use of the term and varied only by such colourful phrases as 'rat poison', 'roach poison', 'corrosive poison', 'deadly protoplasmic poison' and accentuated even by the use of the skull and crossbones as an emblem.

322. Such statements are hardly rational. They reflect merely the intensity of emotion; the fervour that this controversy seems to provoke. They can serve merely to deter by fear, they do not seek to enlighten by information. The fact is that there are many substances essential to human life and nutrition which according to combination or dose are capable of being either beneficial or toxic. A partial list is given in the Ontario report in paragraphs 105 and 106[2]. The fluorides are no different.

323. I am not concerned with elemental fluorine. It is not possible to suggest its association with a municipal water supply. I am concerned with its salts or inorganic compounds. Its numerous organic compounds are not strictly relevant but are considered briefly later (Ch. 42 *post*) for the sake of completeness only.

324. The route by which the fluoride ion is absorbed is via the intestine. The dissociated ion is not absorbed through the skin. The suggestion conveyed to a Hobart audience of citizens by a medical opponent of fluoridation who gave evidence before the Commission, that water fluoridated at 1 ppm might have been responsible for some form of skin irritation or inflammation which he received as a result of dousing his head in a bucket not only lacked contemporary clinical verification but seemed to me in a medical man irresponsible. If it were otherwise, bathing in the sea at 1.4 ppm might be a hazardous procedure.

325. The toxicity of any given dose of ingested fluoride will in the first place depend on the amount absorbed. If taken in solid form, its toxicity will therefore depend on its solubility. Sodium fluoride and other salts which are readily soluble are more freely absorbed than relatively insoluble calcium fluoride, a substantial portion of which will be excreted unabsorbed in the faeces. From the point of view of minimum toxic dosage therefore, the soluble salts are of more importance.

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CHAPTER 19. ACUTE TOXICITY

326. Sodium fluoride is about a thousand times more soluble than calcium fluoride and in the absence of dietary buffers is almost wholly (93% to 97%)[1] absorbed from the gut. It is therefore the obvious case to consider.

327. Roholm[2] reported the smallest single dose of sodium fluoride causing death in the adult to be about 4 gm. Hodge and Smith[3] made a study of the recorded cases of fatalities and cite a dose range of 5 to 10 gm as a reasonable estimate of a ' certainly lethal dose '. In terms of fluoride content these represent approximately a range of 2 to 5 gm or 2,000 mgs to 5,000 mgs or, putting it another way, approximately 30 mg to 70 mg of fluoride per kilo (2.2 lbs) of body weight. Thus by this measure the lowest toxic dose for even a 9 lb baby would be that amount of fluoride, consumed at one time, as is equivalent to that contained in 120 litres or approximately 26¼ gallons of water fluoridated at 1 ppm. The same dose for an adult would require something in excess of 450 gallons.

328. On the other hand, it has been estimated that the maximum non fatal dose may conceivably be as high as 100 gm[4] and doses as high as 23 mg per kg of body weight (i.e. in an 11 stone man a dosage of 1,610 mg) have been given therapeutically for nine consecutive days without signs of poisoning[5]. Other therapeutic doses of from 80 to 400 mg from periods of four to six months have been reported as producing no observable toxicity either clinically or postmortem[6]. More recently doses of 200 mg a day for short term therapy and 40 to 175 mg a day for prolonged therapy up to a year have been employed in the treatment of bone disorders without observable toxic effects[7, 8] and indeed without producing any ' significant alteration in the physiology of any organ except for bone and calcium metabolism '[8].

329. In these circumstances I would adopt the comment by Largent[9] ' It is difficult for persons with only normal imaginations to see any connection between possible acute poisoning by fluorides and the fluoridation of drinking water.'

330. Nevertheless it is suggested by some (certainly the more intelligent of the opponents of fluoridation do not identify themselves with the suggestion) that either by accident or design it is possible for acute mass poisoning of the community to be brought about by the use of fluoridation chemicals or equipment. Though such ideas really belong to the world of fantasy as they are apparently entertained by some members of the community, it is necessary briefly to deal with them. The logistics of such a suggestion are the best answer.

331. It takes 22½ lbs of solid sodium fluoride or 16.7 lbs of sodium silico fluoride in solution to fluoridate at 1 ppm 1,000,000 gallons of water. To do the same for the amount of water consumed in a day in Hobart, say, 10,000,000 gallons, takes 2 cwt or 1½ cwt respectively. To raise that concentration to the level where the minimum fatal dose (2 gms as against a certainly lethal range of 5 to 10 gms)[3] could be consumed in, say, a 10 oz glass of water, it would be necessary to add to the same water supply 700 tons of sodium fluoride or 525 tons of sodium silico fluoride.

332. Hopper capacity is as a matter of design limited to total requirements for one or more days. The concentration achieved by even a week's supply being put into the system all at once (assuming all problems of solubility could be overcome) would still only be about one five hundredth of the quantity required for the minimum fatal dose.

333. Even if one changes the calculation to the amount required for the minimal symptomatic dose of one fiftieth of the certainly fatal dose[10] the margin of safety over normal daily levels is approximately 350 to one.

334. Evidence given by two witnesses as to the fatal effects of an overdose of sodium fluoride when used as a vermifuge in pigs related to dosages of 2 oz of NaF per 200 lb pig, equivalent to .6 gms of sodium fluoride or 600 mgs per kg of body weight. (Though this was the dosage deposed to in the evidence, in fairness to the Department concerned, it was much in excess of the dose recommended in the official publication[11] produced in support.) For a 70 kg man that would be equivalent to a dose of 4.2 gms of sodium fluoride or approximately 20,000 times the quantity

of fluoride contained in 1 litre of fluoridated water. There may be some differences to be pursued between commercial and other grades of the materials used but they do not alter the nature of the illustration. Such matters are not really relevant but are mentioned to dispose of any doubts that may have been engendered by the publicity they received.

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CHAPTER 20. CHRONIC TOXICITY OR FLUOROSIS

Section (a)—General

335. It would be unfair however to identify all the opponents of fluoridation with such ill-considered arguments as are mentioned in the preceding chapter. The main controversy related not to acute toxicity but to the possibility of eventual toxic effects due to the accumulation of fluoride in bones, organs and tissue as a result of prolonged exposure. The term fluorosis or chronic fluorosis is generally used to summarise such effects.

336. Some exposure to fluoride is inevitable in the human environment. It is simply not conceivable that a fluoride-free existence could be the lot of anyone. The question is, what level of exposure to fluoride brings harmful consequences. It is established beyond doubt that the earliest warning and the most sensitive indication of an over-exposure to fluoride is dental fluorosis. The next indication is bony or skeletal changes. These may vary from a symptomless (or even in the aged a beneficial—See Ch. 43 *post*) osteosclerosis or increase in density of bone substance, to abnormal bone growth and ligamentous calcification leading to crippling deformity and even paraplegia due to compression of nerve roots. There are claims of fluorotic changes in other organs and tissues of the body but in my view they are not supported by the evidence as occurring at any level of exposure below that which could be expected to produce symptoms of acute toxicity. Some of these claims I will mention specifically at a later stage but at this juncture discussion of the subject of chronic fluorosis will be confined to the matters mentioned. This is not meant to ignore the topic of abnormal individual reaction, allergy or idiosyncrasy however it be called, which as it bears no relation to the levels of normal variation in community response is, if it exists, unpredictable and unclassifiable. This too will be discussed separately (Ch. 24 *post*).

Section (b)—Relationship between Dental and Skeletal Fluorosis

337. Before reviewing the evidence as to the levels of fluoride exposure at which objectionable dental fluorosis and skeletal changes may be expected to occur, the matter of their interrelationship should be mentioned.

338. The importance of dental fluorosis or mottled enamel as an early warning of a toxic level of exposure to fluoride has not been sufficiently appreciated. It is established beyond controversy that the enamel forming cells of the teeth give the earliest and the most sensitive physiological indication of excessive intake. It is also clear that the warning given by fluorosis in the erupting teeth of children would be manifest at levels of fluoride exposure well below any which had the potential of causing significant skeletal changes in the community and in point of time years before

any such changes could be expected. It is true that dental fluorosis cannot be produced in teeth after the stage of formative calcification has been passed. It ceases therefore to be of any use as an index of fluorosis in the individual after the eighth or ninth birthday. But this does not lessen its value as a community index and if, in the erupting teeth of children, such an indication were significantly to be found, it of itself would be an immediate and unmistakable indication of a community exposure to fluoride which required reduction.

339. It is, as I have said, completely clear that such a warning would be grossly apparent at a level of fluoride exposure much lower and years before symptoms of skeletal fluorosis could be expected in the same community. It therefore, as a sign of over-exposure, constitutes a community warning of great importance, particularly as it lends itself to official surveillance through the school by the agency of the School Dental Service and also because, as will be seen in the next section, the levels of fluoride exposure likely to produce such degrees of dental fluorosis are known with reasonable certainty. In other words, if there is no objectionable dental fluorosis there is no reason to fear that skeletal fluorosis will ever be found in the same community.

CHAPTER 21. DENTAL FLUOROSIS

Section (a)—Fluorotic and Idiopathic Enamel Defects

340. The influence of fluoride on dental structures is most pronounced during the stages of tooth development and maturation, i.e. for all but the wisdom teeth up to the fifteenth year of life. In optimum quantity the fluoride, as we have seen, affects beneficially both the shape structure[1] and enamel of the tooth thus increasing its caries resistance. In the permanent teeth enamel formation begins at birth and should be completed by the eighth year. The first five years of life represent the period of the greatest susceptibility to disturbances of the enamel-forming cells and it is the period most relevant to the ensuing discussion[2, 3].

341. In excessive amounts fluoride may affect some of the enamel-forming cells or ameloblasts adversely. Microscopically such defects appear mainly to affect the organic or interprismatic substance of the tooth. The enamel rods may also show imperfect calcification. In advanced cases the defective development of such areas permits, after eruption, the lodgment of pigments and organic matter in surface irregularities which gives rise to a characteristic staining and discolouration[3, 4]. In milder cases however it manifests as a white opacity against the normal translucent enamel, described by Roholm[5] thus ' . . . the enamel in spots or more diffusely loses its normal translucence and becomes turbid, whitish, with a tone that recalls chalk or unglazed paper '. In such cases the surface of the tooth is smooth and even, without irregularities or defects, and staining does not take place[3, 6]. Where the defect is severe enough to permit of the lodging of pigments, as with all hypoplasias whether fluorotic or not, the discolouration will be progressive[7, 8]. The inorganic mineral of the tooth in such defective areas is not seriously affected but will contain more fluoride[3, 4] than normal enamel. It will be caries resistant. Indeed the whole story of fluoridation derives from the endeavour to understand the reason for what seemed to be a dental anomaly, viz. the high degree of caries resistance possessed by mottled teeth.

342. Mottling or fluorosis of the enamel of erupting teeth may range in degree from grossly disfiguring staining and pitting with brittleness of the enamel surface to the merest white fleck or opacity. The exact mechanism whereby the hypoplasia or mottling is produced is not accurately known, though some causatory factors have been described[6]. This condition in all its manifestations has been loosely called ' Mottling ' and this term is conventionally used in much of the literature. It is however an excessive description of the milder forms of the affliction and the term dental fluorosis is to be preferred.

343. Defects in developing enamel similar to dental fluorosis but due to wholly unrelated causes are common, particularly in Tasmania[9, 10]. It is convenient to refer to them as idiopathic defects to distinguish them from fluorotic. They may be due to a variety of causes, such as vitamin deficiency, trauma, febrile illnesses of childhood and other matters. In recent years the administration during infancy and childhood of the antibiotic tetracycline has been recognised as a potent cause[11, 12].

344. In Tasmania the prevalence and severity of such idiopathic defects in totally unfluoridated areas, such as the Huon and the West Coast[13, 9] has led to suggestions of mineral deficiencies or toxicities[14]. But the Tasmanian picture is typical of what has been reported in low fluoride areas in other parts of the world where reports of children affected range from 83% to 20%

in areas having less than 0.2 ppm fluoride in water[15, 16, 17, 18, 25, 26]. In fact a higher prevalence of non-fluoride enamel hypoplasia in children living in fluoride-free areas than those living in areas with optimum fluoride has been frequently reported and would now be regarded as confirmed[18, 19].

345. In the milder forms their differentiation from fluoride caused defects is difficult[15], even clinically, and it is not always clear whether in fluoridated areas their presence has been sufficiently discounted in investigations as to the amount of mottling which has been ascribed to fluorosis[18]. More careful surveys have in fact shown that, uniformly, fluoride at 1 ppm has led to a decrease both in the prevalence and severity of idiopathic mottling[20, 17, 21, 22]. This has been claimed for Beaconsfield[23] and I accept it. This may be due to the better development of the tooth which an optimum level of fluoride promotes.

346. As a general rule dental fluorosis affects permanent teeth only, though cases in which the deciduous teeth have been affected are reported. These however have only been seen following exposure to fluoride at levels so high as not to bear any practical relation to the matters discussed in this Report. Fluorosis does not develop in erupted enamel[3] though as has been pointed out in cases where the developmental defect is severe enough secondary staining may develop with age at the site of opacities present at eruption.

347. It is the first and most sensitive indication of over-exposure to fluoride. This fact has been used by epidemiologists to measure the limits to which fluoridation should be taken in a community. This has been done by formulating a fluorosis index or measure of the prevalence and severity of mottling in communities whose water supply was naturally fluoridated and by correlating the index thus obtained with the DMF index for the same community and the fluoride level of the water supply. Such indices have been formulated and tested for a number of communities. As it has been well established that the degree of mottling runs parallel to the fluoride content of the drinking water[24] their application to an artificially fluoridated community facilitates the prediction of the amount and degree of mottling that may be expected to follow fluoridation at a given level.

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Section (b)—Classification of Enamel Defects according to Dean

348. The first step in the formulation of a fluorosis index is to establish a standard classification or grading of defect enabling observations by different observers in different areas to be validly compared. As this involves the reduction to verbal formulae of discrete visual impressions it is not without difficulty. Dean, as the progenitor of the fluorosis index, prior to 1942, classified dental fluorosis in six grades according to its severity[1], ranging from questionable to severe. Later in 1942 they were restricted to five, the moderately severe grading being included with the severe, and his classification is now in general use. Dean's own general description of the differences between the gradings thus established is as follows:—

> 'Normally calcified teeth erupt showing a smooth glossy translucent structure, usually of a pale creamy white colour. Teeth affected with a mild endemic dental fluorosis, on the contrary, erupt showing a dull, chalky white appearance; in areas of higher fluoride concentrations the affected teeth may later on take on a characteristic brown stain. In areas of marked severity, the surfaces of the teeth may in addition be marked by discrete or confluent pitting.'.

and his detailed classification is according to the following categories:—

> 'For purposes of recording quantitatively the various degrees of severity the following standard of classification was developed, each tooth present in the mouth being classified under one of the following six groupings:
>
> *Normal.* The enamel presents the usual translucent semi-vitriform type of structure. The surface is smooth, glossy, and usually of a pale creamy white color.
>
> *Questionable.* The enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flakes to occasional white spots. This classification is utilized in those instances where a definite diagnosis of the mildest form of fluorosis is not warranted and a classification of "normal" not justified.
>
> *Very mild.* Small, opaque, paper white areas scattered irregularly over the tooth but not involving as much as approximately 25% of the tooth surface. Frequently included in this classification are teeth showing no more than about 1-2 mm of white opacity at the tip of the summit of the cusps of the bicuspids or second molars.*
>
> * In our earlier studies such teeth were commonly classified as "questionable"; during recent years, however,, they have been invariably listed as "very mild".
>
> *Mild.* The white opaque areas in the enamel of the teeth are more extensive but do not involve as much as 50% of the tooth.
>
> *Moderate.* All enamel surfaces of the teeth are affected, and surfaces subject to attrition show marked wear. Brown stain is frequently a disfiguring feature.
>
> *Severe.* Includes teeth formerly classified as "moderately severe" and "severe". All enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is the discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.'[2].

349. The diagnostic categories so established are expanded in other writings by the same author[1, 3].

350. Such descriptions will convey little, I fear, to the uninstructed. I have had the milder degrees demonstrated clinically and I have seen a number of photographic reproductions, coloured as well as black and white, of all degrees of affliction. I have found uniformly that such photographs are inadequate as an accurate representation of the clinical condition they seek to convey. This applies particularly to the black and white reproductions. They often fail completely to pick out the subtler forms of defect found in the lesser grades, while they can exaggerate grossly the more serious forms. The coloured photographic plates are on the whole better and more accurate. But again they exhibit defects. One is the colour cast associated with colour film and reproduction generally, but more difficult still is the frequency with which the lay observer is misled by light reflections on the glistening wet surface of tooth enamel simulating opacities which in fact they are not. Further they are of course limited to the one plane and generally fail to reveal the condition of the occlusal surfaces, and cusps in particular. These, as Dr Arkle has shown in his most helpful drawings[4, 5] can be quite important in the diagnosis of fluorosis. In the outcome I have decided to reproduce none as part of this Report, though I would mention as probably the most balanced representations the illustrations to an article by Forrest[6].

351. I do however find as a fact that Dean's first two grades, i.e., questionable and very mild, are inconspicuous and would not under ordinary conditions of social intercourse be noticed by any but the professional observer. Whether as some claim the small white opacities which may be found in this condition may be said to enhance the appearance of the tooth as is claimed, is perhaps a matter of opinion but I was struck by the production of artificial teeth (and incisors at that) in which the manufacturers had thought fit to reproduce such an opacity apparently as an embellishment.

352. Further, as to Dean's grades, the comment made by Forrest[7], Diefenbach[8], and developed in evidence by Professors Martin and Storey, is justified: I think that Dean's index is a negative one, an index of defect. He did not take into account as a balancing factor the positive aspect of the improved shape, colour, and lustre which optimum quantities of fluoride will bring in the great majority of teeth exposed to its influence[8]. As a community index of fluorosis it is still in use, but it is not a community index of tooth condition or appearance. For this reason other indices have been suggested[8] but in attempting to cover so much I think they lack the specific quality of Dean's.

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Section (c)—Dean's Community Index of Fluorosis and Its Use

353. In addition to providing a standard for assessing the degree of fluorosis in the individual, Dean (1942)[1, 2] devised means of assessing the prevalence and degree of fluorosis in a group or community. Basically the system which came to be used was to weight the prevalence figure according to its grading for severity by assigning different values for each grade of affliction and the community index of fluorosis was determined by the formula $\frac{\text{frequency x weight}}{\text{number}}$. His own conclusion was that an index below 0.4 was of no public health significance.

354. Dean's observations were made in respect of 5,824 white children aged twelve to fourteen years in twenty-two cities of ten States. Later surveys in other parts of the world have since been published[3, 4]. Dean's observations were reviewed by Hodge in 1950[5] and he demonstrated graphically a linear increase in the severity of mottling when the community index of fluorosis was plotted against the log of the parts per million of fluoride in the drinking water (Fig. IV).

355. When the DMF figures for the same cities were plotted on the same graph, the close relation at 1 ppm between maximum protection with minimum detriment in the shape of fluorosis is demonstrated (Fig. V).

356. In relation to this figure Hodge has been criticised by Dr Exner[6] for omitting reference to the following comment by Dean:—

> 'In your figure one*, I notice you use "very mild", "mild", "moderate" and "severe". I have avoided using these terms as they designate a clinical entity rather than an arithmetically determined community index which I divide as follows according to the index:
>
> | | |
> |---|---|
> | 0.0–0.4 negative | 1.0–2.0 medium |
> | 0.4–0.6 borderline | 2.0–3.0 marked |
> | 0.6–1.0 slight | 3.0–4.0 very marked |
>
> Actually it would make no essential difference in the two lines.
>
> According to my memory, I examined about 2,000 children in five different States with wide differences in the type of mottled enamel before I ventured to set up a tentative classification.'.
>
> * i.e., Fig. I in Hodge's text.

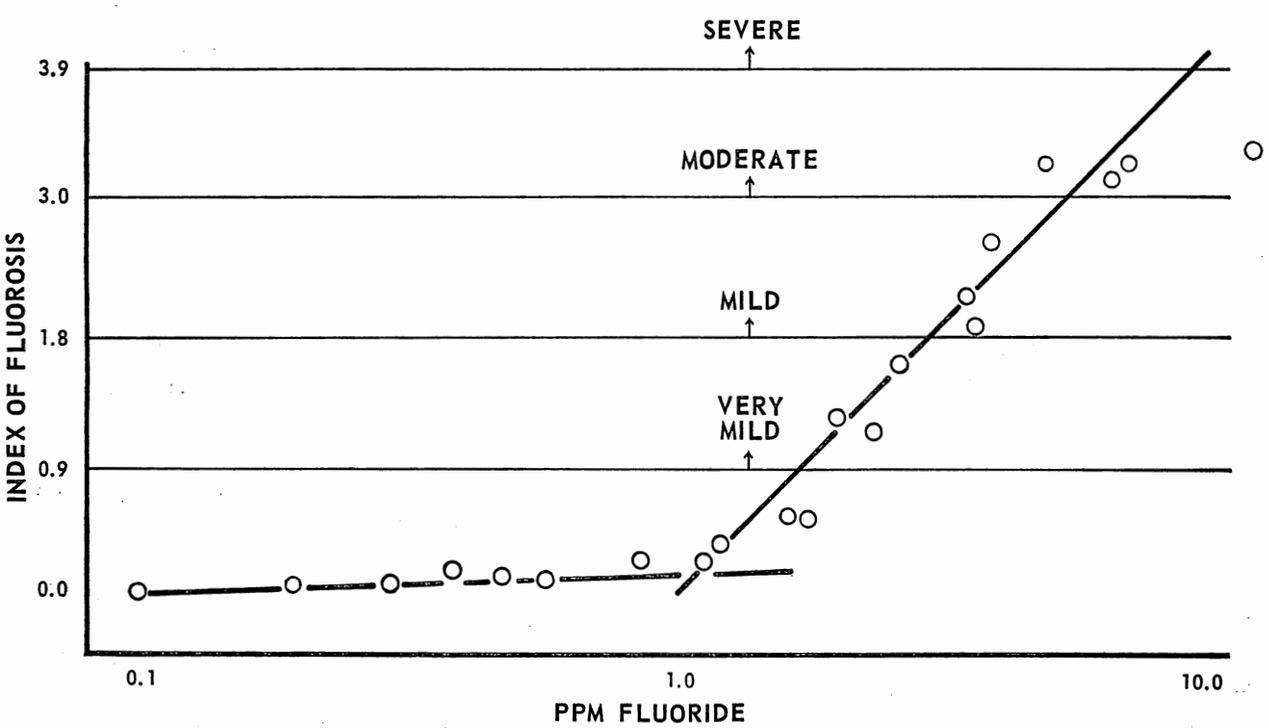
Apart from the fact that Dr Exner has referred to the wrong figure he is technically right in directing attention to the possible confusion that might arise between clinical grades applicable to individuals and a community index. However, it would not confuse one who has followed the controversy. It makes, as Dean says, no difference to the graph and hardly justifies the immoderate manner in which the charge is made.

357. Reviewing the matter in 1963, Hodge[7] says:—

> 'The absence of unaesthetic mottling in communities where the drinking water contains about 1 ppm F and the gradual increase in the number of children so affected as the fluoride concentration increases from 2 to 8 ppm is known quantitatively with a reliability rare in biological data (Dean, Arnold, & Elvove, 1942). The margin of safety is small but is established firmly.'.

FIG. IV

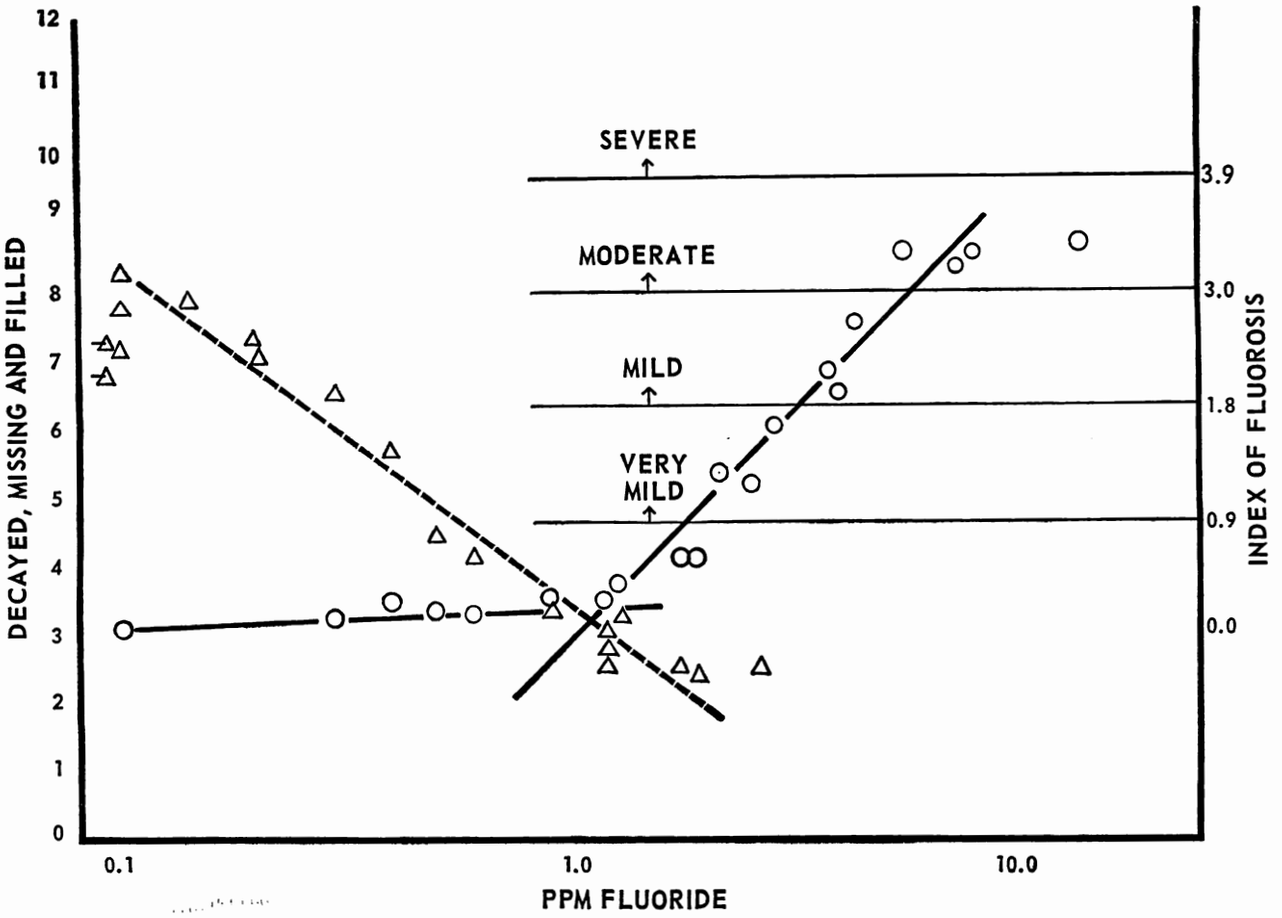
AVERAGE INDEXES OF FLUOROSIS PLOTTED AGAINST THE WATER CONTENT OF FLUORIDE (LOGARITHMIC SCALE)



Taken from Hodge[5].

FIG. V

SIMULTANEOUS GRAPH OF DMF TEETH (DOTTED LINE AT LEFT) AND AVERAGE INDEXES OF FLUOROSIS (TWO SOLID LINES AS IN FIG. IV) PLOTTED AGAINST THE WATER CONTENT OF FLUORIDE (LOGARITHMIC SCALE)



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Section (d)—Effect of Fluoridation at 1 ppm

358. A community fluorosis index does not imply that every child in the community will be affected or affected uniformly. In fact at 1.0 ppm the evidence is clear that at most 10% of children will be affected. From experience gained in fluoridation trials to date the percentage will probably be less. Of those affected it can be anticipated that only some will be 'mild', a few 'very mild' but the majority will be no more than 'questionable'. At such levels, even in the most pronounced cases, the cosmetic defect will be doubtfully apparent to the casual observer without clinical aids, and of very slight consequence if any.

359. But evidence during the last decade seems increasingly to indicate that so far from being the cause of cosmetic defects on a community level, fluoridation will effect an overall improvement. The paucity of fluoride in Tasmanian drinking water has already been referred

to. No accurate survey has been made of the prevalence and severity of idiopathic mottling and hypoplasia on a State-wide basis, but its prevalence is well attested clinically and it has been surveyed and reported as abnormally high in at least three areas of the State[1, 2]. In such areas of Australia as have been adequately surveyed (N.S.W.[3], A.C.T.[4] and Queensland[5, 6]) a high rate of prevalence has been uniformly found and the evidence in respect of Tasmania makes it highly probable that those figures would be at least equalled, if not exceeded. Similar figures have been reported in low fluoride areas in other parts of the world[7] though the Australian figures would seem to be relatively high. In view of the number of well authenticated reports that the intake of fluoridated water at an optimum level appears to reduce the incidence of idiopathic mottling[8, 9, 10, 11, 12] I would confidently expect that a similar intake in Tasmanian communities, quite apart from all questions of shape and colour of teeth, will effect an overall improvement by reducing the frequency of idiopathic enamel defects which would otherwise be found.

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Section (e)—Some Reports of Fluorosis at Lower than Optimal Levels

360. Some attempts were made to suggest the likelihood of a higher prevalence and degree of mottling as likely to result from fluoridation at 1 ppm than would be indicated by the preceding considerations. Some of these attempts were rather crude[1, 2] indicating some shuffling at least of figures and photographs to suggest alarming degrees of fluorosis[3] as likely to result from fluoridation at 1 ppm. In the documents mentioned, by marrying photographs taken in a survey of the city of Bauxite (13.7 ppmF) to the degrees of mottling found in Newburgh and Grand Rapids and by suppressing the actual detailed description of what was in fact found in those two places and which went to contradict any impressions that might be gained from the photographs as in any way typical of the places mentioned, the effect was plainly to suggest something which the facts do not warrant. The matter is not helped by such examples and they are to be regretted. Another argument involved the use of the 'questionable' category to produce percentages of total mottling notwithstanding that in the cases referred to the weighted fluorosis index for the community was well below accepted levels. I make no special mention of the fact that in the one example in which I sought to check the figures cited from the report from which they are supposed to be extracted I could not get coincidence in a single instance[1, 2, 6]. It may not affect the overall argument but it is rather curious. I have been unable to find a reason. Other references in the same and similar documents are to some of the Arizona cities studied by Galagan[38] as a result of which the relationship of fluoridation levels to high temperatures is delineated, but this factor is not allowed for.

361. Confusion is possible. The results for both Newburgh-Kingston and Grand Rapids have been cited by both sides but different interpretations put upon them. To me it is clear that the category of 'mild' mottling is the only one worth bothering about. 'Questionable' is, as the name implies, a clinical question mark; in Dean's words 'the experienced investigator occasion-

ally hesitates to classify either as apparently normal or very mild.'[4] and ' very mild ' is completely minimal. I am satisfied neither affect adversely the function or the aesthetic appearance of the mouth or are capable of doing so. ' Mild ' as a classification is warranted on Dean's classification where in at least two teeth ' The white opaque areas in the enamel of the teeth involve at least half of the tooth surface. The surfaces of molars, bicuspids, and cuspids subject to attrition show thin white layers worn off and the bluish shades of underlying normal enamel. Light brown stains are sometimes apparent, generally on the superior incisors.'[4].*

* Later Dean modified this definition as indicated in paragraph 348, *supra*. The earlier definition is applied in the Newburgh-Kingston study[5]; the later in the Grand Rapids study[6].

362. In the Newburgh trial there were six cases of mild mottling found out of 438 children, i.e., 1.3% after ten years. The examiners' remarks were as follows[5]:—

> ' The distinctions between questionable, very mild and mild fluorosis are based wholly on the extent of the tooth surface involved. In these three categories noted, the involvement consists of slight whitish flecks or opaque areas on the surface of the enamel without any breaks in surface continuity, excessive wear or staining. A trained eye is required to detect these stages of mottling, and the average dental practitioner would have difficulty finding them. None of these instances of dental fluorosis was disfiguring.'.

As against this there was a significant decrease in non-fluoride opacities as against the control city of Kingston:—

> ' In Kingston 115 of the 612 children examined showed nonfluoride opacities, constituting almost 20% of the children examined. In Newburgh there were thirty-six such children, or 8% of the children studied. Most instances of these hypoplasias appear as circular white or colored patches in the enamel, and most of them are obvious even to the untrained eye. The relatively infrequent occurrence of nonfluoride opacities in Newburgh compared to Kingston tends to confirm a previous report that the intake of fluoridated water at the recommended concentration appears to reduce the hazard of hypoplastic spots on teeth.'.

363. In Grand Rapids after seventeen years of fluoridation, out of 358 children both negro and white examined with continuous residence there were six cases of mild fluorosis or 2%. The examiner's (Russell) comment was[6]:—

> ' The few teeth characterized as having mild fluorosis were near the minimum limit for this category. None of the fluorosed teeth was stained and none was obvious enough to impair the appearance of the examinee in smiling or in speech.'.

He further reported:—

> ' 3. Pigmented enamel blotches due to causes other than fluoride were somewhat less prevalent in children with continuous residence than in children who had spent some part of their lives in some other place.
>
> ' 4. These findings support the estimate that, at 1.0 ppmF in the community water, there is a twofold margin of safety against mottling.'.

Such figures and comments do not in my opinion justify the fears that documents such as Ex.494[1] (a pamphlet circulated in Victoria headed ' Who has the right to inflict such ugly teeth on other people's children ') seek to arouse or the uses made of such a document.

364. There is however a theoretical risk which I do not overlook that in these few mild cases a few may develop some light staining later in life. It was not found after seventeen years in Grand Rapids and has not been demonstrated in any of the other artificial fluoridation trials that have been brought to my notice. Its possibility has to be offset against the stronger probability that over the child population as a whole there will be a significant lessening of hypoplasia due to idiopathic causes which can become pigmented just as readily but which preferentially is found on the anterior teeth and hence is more noticeable.

365. I should add that in the clinical examinations conducted in Beaconsfield in my presence, ' very mild ' was the highest category found and the prevalence was well within expected limits. At the same time the clinical evidence is there has been a noticeable decrease in idiopathic mottling which is in accordance with expectations[7].

366. Dr Sutton, however, referred to some papers in which examples of dental fluorosis, exceeding in degree of severity those that might be postulated on Fig. IV (*supra*)[8] had been found in communities whose naturally fluoridated water was either below or only slightly above the optimum level of 1 ppm. Such reports do exist but, accepting their accuracy— and I think there are substantial reasons for doubting the data in some of them—I am satisfied they are

clearly distinguishable and have no application to Tasmanian conditions. They relate to arid tropical or sub-tropical zones, e.g. parts of India, Israel[9] and Arabia where the temperature range and dietetic habits are quite dissimilar to those prevailing in this State. The Indian studies of Singh and others will be discussed in detail in the next chapter but in relation to them I think it is clear that not all dietetic factors have been adequately taken into account in assessing the total fluoride intake.

367. Another example cited by Dr Sutton[11] and others is a 1941 paper of Dr Sognnaes in relation to the inhabitants of Tristan da Cunha[10]. He reported a condition " suggestive of threshold dental fluorosis ' notwithstanding that the ' common drinking water is relatively soft, and that its fluorine content (0.2 ppm) is below the amount (1 part per million) which is considered causative of clinical changes in developing enamel '. The same paper was relied on by Dr Exner[12]. This observation has long been a subject of debate. Subsequent writers have drawn attention to the diet of the islanders high in fish and other marine products. Minoguchi, whose Japanese studies in relation to dietary fluoride make his views particularly significant, said in 1967 :[13]

> ' Up to now, only one investigation report (Sognnaes, Tristan Da Cunha Island) has stated that dental fluorosis may be caused by fluorine contained mainly in food. This report is a special case under a special life environment.'

But there is considerable doubt about the whole matter and I note that the South African Commission took evidence from Dr T. Ockerse who had visited the island. He (and his name is a familiar one in the literature) denied in evidence that the condition described by Sognnaes was caused by fluoride at all and his evidence was accepted[14]. Professor Steyn[15] also refers to Ockerse's observations. Following the matter still further, it hardly seems that full justice has been done to Sognnaes himself. He reported ' white spots ' on both deciduous and permanent teeth and that they were most frequently seen on the anterior teeth. These matters are so atypical of fluorosis at low levels as of itself to make the diagnosis doubtful. It appears to have been no more than a speculation on his part and he himself entertained the possibility from his data that factors other than water fluoridation might have been responsible[16]. In sum the suggestion is strong that this was a mistaken diagnosis or perhaps an over-statement based on a doubtful or provisional diagnosis at best. This would seem to be confirmed by more recent work[16, 17].

368. Another early report of the same nature cited by Dr Sutton, who did not however refer to the later work in which it has been criticised, was a report by Kemp *et al* (1942)[18] of severe dental fluorosis in a village of Oxfordshire with 8 ppmF. In fact the condition does not seem to have been fluorosis at all but a hereditary complaint[19]. Other aspects of the same work relating to skeletal fluoride were not confirmed by later work[20].

369. Still another early report by Dr H. V. Smith[21] which was urged on me to the same end must be read subject to the later qualification expressed by its author in a communication made to the New Zealand Commission, the effect of which is to remove the report from the area of controversy[22].

370. An English study which is often quoted and which is worthy of more attention is that by Weaver (1944)[23]. This should be read with a later paper[24] in which some modification (supporting the fluoride hypothesis) of the earlier views is evidenced and admitted. It is of interest as relating to an English community where some dietary habits at least (e.g. tea drinking) might be thought to be paralleled in this country and as the article indicates, the standard of nutrition generally was rather low, thus providing a rather severe test. Two adjoining communities were studied, North Shields (less than 0.25 F) and South Shields (1.2 to 1.8 F). After noting that in South Shields generally speaking the mottling was ' not at all obvious ' he reported:

> ' Of the 500 South Shields children, twenty-eight were recorded by me as questionable cases of mottling, a paper-white patch suggestive of mottling being observed on one tooth only. I regarded eighty-eight children as showing definite mottling of very slight degree and eighteen others as of slight degree. Paper-white areas involving less than 25% of the tooth surface were the criterion for inclusion in the ' very slight ' category. When 25-50% of the tooth surface was involve d, the child was recorded as having " slight " and " slight " cases represents 21% of the children. Only two cases were seen with brown pigmentation of the mottled teeth, and in both cases it proved possible to examine a younger sister aged ten years. In one case the younger sister had a perfect set of teeth without a trace of mottling; in the other case there was one well-marked mottled but not pigmented patch on a lower premolar . . . Of the 106 children classed as having mottling, only forty-seven showed any trace of the condition in the incisor teeth. . . . .

Amongst the North Shields children, thirteen were recorded as questionable cases of mottling and two others as " very slight[23]".'

In an area in which the level of fluoride was above the optimum (the water being drawn from a dozen wells ranging in fluoride content from 1.2 to 1.8 ppm) pigmentation was seen in two out of 500 12-year-old children. The teeth affected are not stated. The reference to the North Shields children makes it unclear as to what effort if any was made to distinguish between fluorotic and idiopathic opacities. I see no discrepancy between this early report and the conclusion being discussed. As Weaver himself said in the later paper:[24]

'My own findings in South Shields fitted in very well with Dean's figures.'

371. Another finding by Weaver in relation to the same study is not without interest. Assuming all his mottling in South Shields to be fluorotic—and he makes no distinction—he found no relationship between mottling and caries incidence, i.e. no greater susceptibility to caries in fluorotic teeth than in others. He said:

'The South Shields children who showed no mottling had on the average substantially the same number of carious teeth per child as was found in the children who showed mottling. It has been suggested that in children whose nutritional condition is subnormal, mottling tends to be accompanied by pitting and attrition of the enamel but in South Shields no case of pitting or attrition was seen although almost 13% of the children were regarded as nutritionally below normal[25].'

Perhaps in this connection a later English study by Forrest (1956)[26] should be looked at. A survey of mottling in various areas with water ranging in fluoride content from 5.8 to 0.1 gave the results shown in Table XVIII. It will be noted that in relation to Slough one case of 'moderate' is recorded. It could be this is due to the fact that according to the text she has modified Dean's definition of 'mild' and 'moderate'. 'Cases were counted as "moderate" if there was any brown staining at all even without white mottling, providing that it did not involve more than a quarter of a tooth's surface.' Many such cases would, under Dean's classification, be regarded as 'mild', so that the comparison ceases to be precise. However this is unusual, even atypical. The text says of Slough:—

'In Slough (0.9 ppm F) "questionable" or "very mild" mottling occurred in 16% of the children but its detection required very careful examination. Mottling which might be termed obvious was found in only 3% of the children.'

TABLE XVIII

THE INCIDENCE OF VARIOUS TYPES OF ENAMEL OPACITIES IN CHILDREN AGED 12-14 IN FLUORIDE AND NON-FLUORIDE AREAS

Area	Percentage of Children with Enamel Opacities									Index of Excluding idiopathic spots	Mottling† Including idiopathic spots
	Fluoride content of water p.p.m.	Number children examined	No opacities	Idiopathic spots only	Degree of mottling: Not noticeable		Noticeable				
					Questionable	Very mild	Mild	Moderate	Moderately severe		
West Mersea ..	5.8	51	0	4	16	12	2	25	31	2.5	2.6
Burnham-on-Crouch	3.5	62	5	3	10	29	16	26	11	1.88	1.9
Harwich	2.0	92	18	16	22	32	4	8	..	0.7	0.8
Slough	0.9	119	50	31	10	6	2	1	..	0.17	0.32
Saffron Walden and District ..	0.1	145	37	30	*12	*9	*6	*6	..	*0.45	*0.6
Surrey Schools ..	0.1–0.2	114	40	30	*14	*3	*2	*11	..	*0.45	*0.6

*Not due to fluoride ingestion, but graded as if they were.

†By Dean's method of computation. The threshold of objectionable mottling lies between 0.4 and 0.6.

Taken from Forrest (1956)[26]

372. Another argument was based on reports of dental fluorosis at suboptimal levels in cases of severe malnutrition[27]. The precise correlation here is not clearly demonstrable though there is some evidence that a lowered vitamin intake particularly of vitamin C may be implicated[28, 29, 30]. To guard against the possibility that there should be some significance in these reports, I caused them to be referred for evaluation to witnesses familiar with Australian and Tasmanian dietary standards and habits with negative results. Again in the face of the overwhelming evidence of similar effects not having been observed in temperature zones of adequate nutritional status, I

can see no immediate relevance in such studies. Obviously such matters should be kept under review. It may yet be the case that specific dietary deficiencies as distinct from the general and severe deficits that have so far been demonstrated in the cases referred to will be pinpointed by further study but such reports do not constitute a present objection to fluoridation in this community.

373. The further claim is made that 'mottled teeth are somewhat more resistant to the onset of decay, they are structurally weak; when decay does set in, the result is often disastrous[31, 32, 33].' This is not a precise statement and in its imprecision it is misleading. No attempt is made to indicate the degree or grade of 'mottling' to which such a condition is attributed or the level of fluoridation implicated except that in one early article by H. V. and M. C. Smith a level of 1-6 ppm is indicated (stated as 1.6 to 4 ppm in a later paper)[32]. These are the same authors whose communication to the New Zealand Commission modifying their early viewpoint I have already referred to (§ 369 *supra*). Precise studies at optimum levels have indicated the reverse. Thus Backer Dirks[34] '. . . . the speed at which cavities increase in size is less in the fluoridated area' and the enamel in the 'white spot cavities' of incipient caries in fluoridated areas is less soluble than intact enamel from the same teeth[35] due, it seems, to its capacity to attract and concentrate fluoride in the process of remineralisation.

374. At levels of fluoridation at which severe mottling may be found the teeth become excessively hard and brittle. This makes them difficult to treat because of the possibility of fracture. The matter was fully reviewed in the evidence by Professor Martin whose evidence I accept. The claim has no relevance to fluoridation at 1 ppm. Incidentally I notice that the British Dental Association[36] in a pamphlet for popular consumption has commented on the matter thus:

> 'Q. *Does fluoride so affect the teeth that they cannot be filled satisfactorily?*
>
> 'A. Excessive fluoride intake, such as may occur in a number of cases when the fluoride content of the water is 4 ppm or more, will produce dental enamel which is so defective that it will chip and flake off making it difficult to obtain a satisfactory enamel edge for a filling. The beautiful enamel, such as is found for instance in Slough, Bucks (0.9 ppm F), presents no such problem. Drs H. V. and M. C. Smith are quoted as having said that decay in fluorine-containing teeth is exceptionally severe and difficult to repair and that when decay sets in the result is disastrous. When they made this statement they were not satisfied that an appropriate concentration of fluoride in a water supply could be determined and maintained. They have since expressed their satisfaction that this can, in fact, be done and have withdrawn their objection.'.

There is authority for the last sentence[37]. It conforms with the communication reported by the New Zealand Commission to which I have already referred. I have not thought it worth while to pursue the matter further. The rest of the paragraph accords with my understanding of the evidence.

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Section (f)—Dental Fluorosis—Conclusion

375. However the real nub of the objections raised under this head is that fluoridation at the optimum level of 1 ppm, being a compromise between maximum protection and minimal detriment, necessarily accepts the fact that there will be some dental fluorosis or mottling in the teeth of some children who might otherwise not have had it. The argument tended to become emotional. Mottling is too wide a term. Because technically it can be used to cover the pitted and stained and undoubtedly repulsive teeth of severe fluorosis, it is difficult perhaps to avoid emotional overtones in any reference to the subject. In studies of artificially fluoridated areas I am not aware of any investigation having reported a higher degree of fluorosis than 'mild'. (Cf. Maier (1963)[1] 'not a single such instance has so far been observed') and these are few. I have referred to two of the most prominent studies already. In neither of these did the prevalence exceed 2%. I could add other examples, e.g. Brantford[2]. In these cases this 'mild' classification reflects only a greater degree of involvement of the surface of the teeth with a minimum of two. It does not reflect the further fact that in the lower grades of fluorosis with which we are here concerned 'opacities occur symmetrically, mostly on the premolars, the cusps of the permanent molars and, to a lesser extent, on the incisors . . . Idiopathic opacities on the other hand, . . . are most commonly found on the first permanent maxillary incisors'[3, 4]. The latter are of course the teeth most subject to the scrutiny of the casual observer. Under the conditions of artificial fluoridation no investigator has reported disfiguring fluorosis. Indeed most specifically report the opposite. None has been reported found in Beaconsfield after fifteen years of fluoridation[5].

376. The theoretical risk that any hypoplastic area may subsequently become pigmented in adult life[6] is undoubted and this is a factor to which perhaps too little attention has been paid. Its likelihood depends in the first place on the severity of the initial enamel defect[7, 8, 9] while the degree of aesthetic objection will depend on its site in the mouth. If anterior teeth are involved, and these are involved less frequently than others, it could become a matter of importance. Dr Sutton agreed the total incidence of 'mild' with optimum fluoridation would be no more than 2%[10]—it could in my opinion be less—and only some small portion of this percentage would be theoretically at risk. The probabilities of this concurrence of conditional events, in a class of no more than 2% is hard to assess, harder still to express quantitatively. I can only report that I do not regard it as a serious risk and I have yet to see or read of it demonstrated in practice rather than theory. On the other hand I am satisfied that, if the overall reduction of idiopathic mottling, that I would be confident in anticipating, be taken into account, the net gain is unquestionably and substantially in favour of fluoridation.

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CHAPTER 22. SKELETAL FLUOROSIS

Section (a)—Critical Levels of Exposure

377. Skeletal fluorosis has been extensively studied. The monograph of Roholm (1937)[1] on the health status of cryolite (sodium aluminium fluoride) workers in Denmark is usually taken as a starting point. Under the industrial processes then in use, workers in Copenhagen had been exposed for many years to heavy concentrations of fine dust with an average content of 97% cryolite. The skeletal and other disabilities which resulted led to his investigation which is still regarded as a model of its kind. Since then conditions of skeletal fluorosis have been described and documented in many parts of the world, both in men and in animals.

378. Two stages are recognised. The first is an asymptomatic increase in bone density on X-ray (osteosclerosis) with a tendency to bony outgrowths and calcification of ligament attachments. Changes are usually seen first in the pelvis and lumbar spine. The second stage is symptomatic—'crippling fluorosis', 'poker back'. There is stiffening of the back and in severe cases, restriction of respiratory movement and neurological complications due to compression of nerve roots.

379. However such changes are not in themselves pathognomonic of fluorosis but may be found in many diseases and conditions unassociated with fluoride[2] and the diagnosis of fluorosis must always remain uncertain unless bone or urinary specimens reveal evidence of high intake and a history of excessive exposure can be obtained. Largent[2] suggests that the corroborative evidence required for a diagnosis of moderate or advanced fluoride osteosclerosis should be (*a*) proven absorption of fluoride amounting to 10 or 15 mg a day or (*b*) urine concentrations of fluoride averaging more than 8 mg per litre over a period of more than five years. In relation to bone fluoride content the critical figure is suggested by Jackson and Weidmann as being between 2,000 and

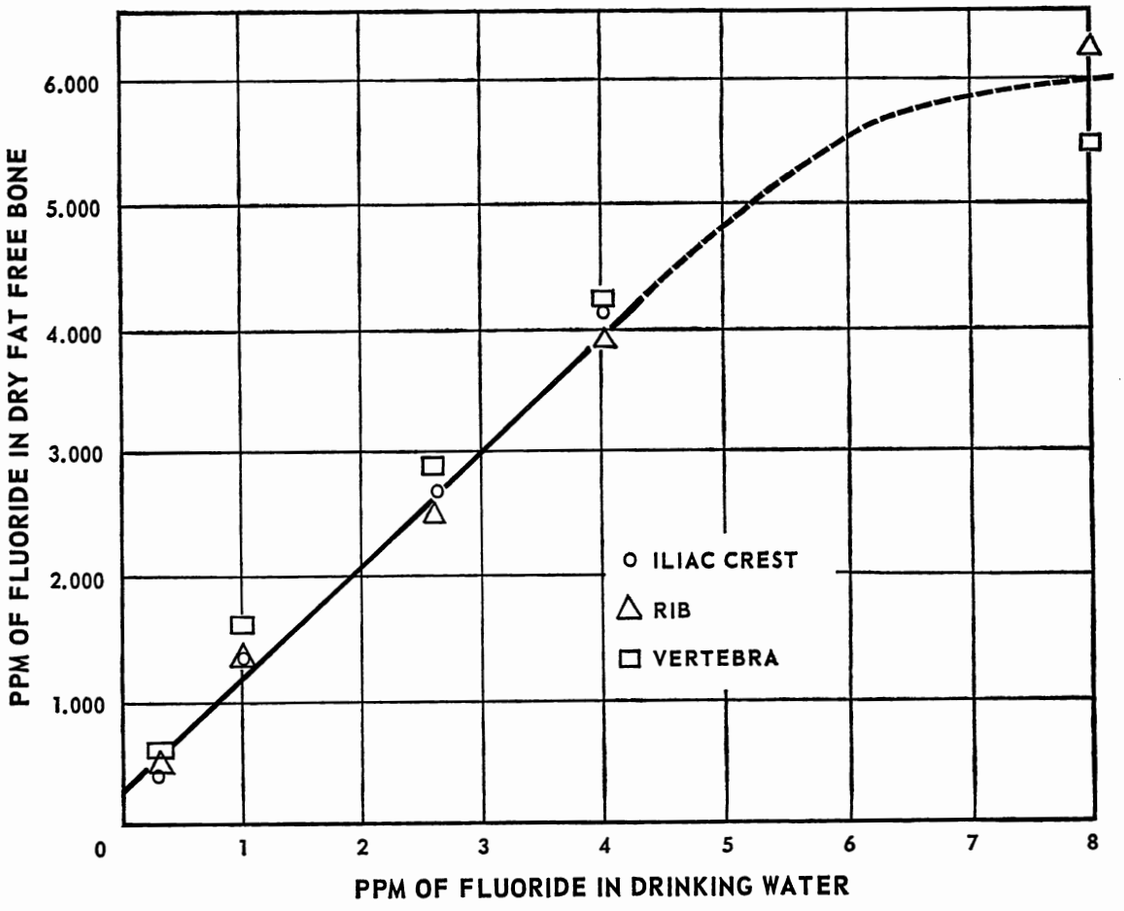
3,000 ppm fluoride in dry fat free weight in compact bone and 4,000 to 6,000 ppm in cancellous bone[3]. This, it is claimed, agrees well with McClure's index of 5,000 to 6,000 ppm dry fat free bone[4]. However, such values have been frequently recorded without detectable changes in bone structure[5, 6].

380. These are high values. I know of no study nor any report which would suggest that they can be approached in normal individuals in temperate zones from drinking water fluoridated at 1 ppm. Weatherell[7] reviews the literature and concludes that below 4 ppm even life-long ingestion seems to have no deleterious effects. This is confirmed by Singh and Jolly[8] and accords with the data established by McClure (1958)[4] and Zipkin (1958)[9] in their studies of the correlation between fluoride in bone and in the drinking water. F. A. Smith[10] has combined the results of their investigations in the one graph which I reproduce (Fig. VI). It relates to persons whose ages ranged from twenty-six to ninety years and whose use of their respective water supplies varied from ten to eighty-seven years. These are minimum levels. Other studies have suggested they should be higher[11].

381. As to crippling fluorosis all agree that continuous exposure to fluoride which results in an intake of 20 to 80 mg a day for ten to twenty years will ultimately produce skeletal fluorosis in a severe form. As against that the adult male intake of fluoride from all dietary sources in temperate zones in a tea (and beer) drinking community with water fluoridated at 1 ppm has been estimated as no more than 3.2 mg daily[12, 13]. The argument has therefore been whether the lower limits of the range suggested, i.e., 20 mg a day for ten years, are in fact threshold values and whether some individuals in the community might not, by reason of disease or dietary habit, achieve a total intake which brings them within the danger range even with water fluoridated at 1 ppm.

FIG. VI

RELATION BETWEEN FLUORIDE IN DRINKING WATER AND FLUORIDE IN HUMAN BONE



Taken from Smith[10] and compiled from data of Zipkin *et al.* (1958)[9] and McClure *et al.* (1958)[4].

382. The exact minimum level of fluoride intake which will result in symptomatic skeletal fluorosis is not established. The South African Report observes that the ethic against human experimentation may preclude such information from ever becoming available. But the evidence against the possibility that such a result could ever be achieved in a temperate climate at 1 ppm is both theoretical and epidemiological. As to the first I quote from Hodge and Smith[14]:—

> 'The question as to what fluoride intake is responsible for the development of crippling fluorosis cannot be answered with specific numbers. The guess made some years ago that 20 to 80 mg fluoride or more per day must be absorbed to produce crippling fluorosis in ten to twenty years is still a valid one. A prediction can be based on the bone fluoride concentrations reported by Roholm: rib and vertebral concentrations of fluoride ranged as high as 9,000 to 13,000 ppm. Taking the data of Zipkin *et al.* (1958b) on the bone fluoride concentrations of individuals who had lived all their lives in areas where the water fluoride content was known (up to 8 ppm) and extrapolating, a rough prediction can be made that if an individual drank water containing 20-24 ppm (other factors remaining constant), concentrations up to 13,000 ppm of fluoride would ultimately appear in the skeleton. Since the average person probably drinks about a liter of water a day, this corresponds to fluoride absorptions of at least 20 to 24 mg fluoride, surprisingly close to the estimated minimal dust exposures. Even though the amounts to produce crippling fluorosis are not precisely known, it is generally accepted that this disorder develops with exposures much greater than those which will produce osteosclerosis in five to ten years. It is thus necessary only to ensure that exposure conditions are such that the urinary excretion never exceeds 5 mg per liter thereby forestalling osteosclerosis and preventing beyond doubt the development of crippling fluorosis.'.

383. The constancy of the relation between the urinary excretion rate and the fluoride level of the drinking water in constant use where a physiological balance has been achieved is well attested[15]. Thus, to achieve the constant urinary rate of 5 ppm over a period of years postulated by Hodge and Smith would certainly postulate on all available evidence a fluoridation level of not less than 4 ppm.

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Section (b)—Epidemiological Studies

384. As to the epidemiological evidence, the main controversy has, both in the evidence and in the literature, centred around what is known as the Bartlett-Cameron Study[1, 2]. In this study the results of detailed medical examinations made at ten years' interval of 116 persons in

Bartlett (with natural fluoride at 8 ppm until eighteen months before the end of the ten-year period when it was reduced to 1.2 ppm) were compared with 121 in Cameron (fluoride at 0.4 ppm). All were chosen at random from persons who had resided in their respective communities for at least fifteen years. Ages ranged from fifteen to sixty-eight in 1943 and, by the time of the second examination in 1953, the average period of exposure was 36.7 years. It was claimed that no significant differences were found between the results in the two towns except for a slightly higher rate of cardiovascular abnormalities in Cameron and a marked predominance of dental fluorosis in Bartlett.

385. This study is heavily relied on by the proponents of fluoride. It was cited and its conclusions used by the Research Committee of the United Kingdom Ministry of Health in 1962[3] and by the Expert Committee of the World Health Authority in 1958[4]. Elsewhere it is claimed that it provides 'the strongest evidence against such dietary fluoride having any deleterious effects' but it has been just as heavily assailed[5, 6, 7]. Broadly speaking the complaint is that the sample populations (116 in Bartlett, 121 in Cameron) were too small and that too many variables and extraneous factors have not been accounted for in the conclusions which were drawn.

386. The criticisms reflect the difficulties of conducting such studies. It was sought to make a comparison over ten years of matched population groups between a high and a low fluoride area with a view to determining whether any significant health differences could be discerned which might be attributed to fluoride (at 8 ppm) in Bartlett. In such a study over a ten-year period, changes in the groups studied in sex and age distribution and total number would be inevitable due to death, change of residence, etc. Although 'removed' residents were followed up as far as possible, nevertheless the fact of their removal necessarily varied the fluoride exposure. Further, the study was handicapped by the fact that eighteen months before the end of the trial period the Bartlett water was defluoridated from 8 ppm to 1 ppm. This had the effect of setting up a negative fluoride balance in the inhabitants of Bartlett which continued for at least two years and led to fluoride being mobilised from the bones and excreted in the urine[8]. I am uncertain how far this could be expected to reverse any gross bony changes if previously present. Roholm thought it possible even probable[9] but it is a subject on which precise data would seem to be lacking. However these defects in the conditions of the study are undeniable and obviously they impair the conclusive nature of the statistics produced.

387. But not enough credit is given for the extensive nature of the medical, radiological and pathological data which the authors of the study set out to collect and the thoroughness with which the medical status of the individuals examined was explored. One must refer to the studies themselves for the full details. Both in 1943 and 1953 they included a medical history, physical and dental observations, X-rays, blood and urine studies. In the later study[2] detailed comparisons of bone densities and conditions were made in respect of the same patients as were comprised in the first study. The results showed evidence of increased bone density or an asymptomless osteosclerosis in the high fluoride area in 10 to 15% of the cases studied with no deleterious bone changes and no associated ill effects. The changes seen were slight, often equivocal, bore no resemblance to the conditions described by Roholm and could not be ascribed to fluoride alone in view of the fact that some of the same symptoms were found in the non-fluoride area and are often associated with senile changes in such areas.

388. Jenkins (1955)[10] while agreeing that statistically the numbers involved were too small to establish conclusively the negative proposition which it sought to examine considered 'that it might be adequate to demonstrate a difference if it existed.' But in this context at least, the real point is that in this series of careful and thorough examinations, with the exception of dental mottling, nothing approaching the symptoms of disabling fluorosis as described by Roholm and others can be discerned. This in a hundred or more patients selected at random and examined at ten-year intervals with a fluoride exposure of up to 8 ppm over a period averaging thirty-seven years. While it may lack conclusiveness as an exact study of the relationship between fluoridation at 8 ppm and 0.4 ppm, to deny it any significance or relevance to an inquiry as to the results reasonably to be expected with fluoridation at 1 ppm is in my opinion to be captious and unreasonable and I so report.

389. But few, if any, epidemiological studies can or should be expected to stand on their own. The point is well made by Martin[11]. The criticisms made of the Bartlett Study do not establish a negative. Though attempts have been made to establish a significant difference in the mortality rates of the two cities, corrected for age they have not been demonstrated as statistically significant and all subsequent evidence serves not to contradict but to corroborate.

390. Specifically I would refer to the following studies which I extract from a fairly copious literature:—

(*a*) Stevenson and Watson (1957)[12] in 170,000 X-ray examinations found increased bone density in twenty-three patients only. All twenty-three lived in areas where the fluoride content of the water ranged from 4 ppm to 8 ppm. In only one of the twenty-three cases was it as low as 4 ppm. No systemic effects and no complaints were related to the bone conditions observed.

(*b*) Eley *et al.* (1957)[13] in examinations of 328 boys from high fluoride areas (1.3-5.8 ppm) and 352 from low fluoride areas (0.1 ppm or less) found no difference in spinal diseases or defects.

(*c*) Geever *et al.* (1958)[14] found no significant findings in 728 post-mortem examinations of residents of Colorado Springs (2.5 ppmF) either on the basis of intra-group comparison between those with over twenty years' residence and those with less or in a comparison with statistics related to other population groups.

(*d*) Jackson and Weidmann (1958)[15] in a paper already referred to (para. 379, *supra*) compared bone specimens obtained post-mortem from West Hartlepool, South Shields and Leeds and concluded that artificial fluoridation at 1 ppm does not enable bone to accumulate fluoride to a critical level even in tea drinking communities.

(*e*) Knizhnikov (1958)[16] in a detailed controlled study between two Russian towns, one with water fluoridated at 4 ppm, the other with 0 to 0.9 ppm, concluded that apart from mottled enamel there were no significant differences between the health status of the two towns.

(*f*) Zipkin *et al.* (1958)[17] made a systematic survey of bone fluoride content and the bone histology of sixty-nine persons who had used water containing fluoride in concentrations ranging from 0.1 to 4.0 ppm for ten to eighty-seven years. Where water was 1 ppm the bone averaged 1,380 ppm in dry fat-free bone (2,250 in ash) and where it was 2.6 ppm it contained 2,670 ppm and 4,500 respectively. All bones studied were microscopically and macroscopically normal.

391. There are others but these will suffice. On this evidence and in the light of the discussions with the witnesses who gave verbal evidence and my own reading, I have not the slightest hesitation in adopting the finding of the South African Commission[18]:—

> 'It is abundantly clear that the consumption of water fluoridated at 1 ppm for a lifetime will not lead to the accumulation of harmful concentrations of fluoride in bones and even higher levels of consumption (2.6-4.0 ppm for many years) could not be expected to produce deleterious effects.'.

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Section (c)—Skeletal Fluorosis in Native Populations

392. I do so despite studies of endemic skeletal fluorosis as found in native populations of India, Arabia, China, Algeria and South Africa which might superficially be thought to require a modification of this conclusion. Their evaluation is difficult because of dietary and nutritional differences, unusual environmental factors, such as temperature, doubts as to analyses and unsatisfactory water histories.

393. The studies most frequently cited are those of Professor Amarjit Singh and his colleagues of the Medical College, Patiala, Punjab, India. Their investigations up to 1962 are summarised in a chapter[1] headed 'Endemic Fluorosis' contributed by Singh to the papers of a Berne Symposium of that year. However, as this summary is often deficient in detail it is necessary to refer to the earlier papers[2, 27, 28, 29] for information on some matters of importance.

394. The papers survey a number of cases of fluorosis from two districts of the Punjab in which fluorosis is heavily endemic. The area surveyed has a fairly hot summer, the temperature touching 116° to 120° F and has a dry and sandy soil. The chief sources of drinking water are artificial wells and the fluoride content varies from 2 to 18 ppm.

395. Four hundred and nine cases of endemic fluorosis were seen over a period of seven years. Most were studied in the field, i.e., clinically but forty-six were hospitalised and a comprehensive study made according to fixed plan. I quote the description of the method followed[2]:—

> Every tenth house was surveyed, and adults over 30 were scrutinised for bony exostoses, stiffness, and limitation of movements in the spine and other joints, and evidence of dental fluorosis. Radiographs of all cases with positive clinical features were taken with a standard exposure. Water and soil samples from different parts of this region were analysed for fluoride content. The results varied, but the fluorine content of the drinking water was uniformly high—as much as 14 parts per million in places.'.

396. It is apparent that the main purpose of the paper is to report an epidemiological survey of skeletal fluorosis with and without neurological symptoms. Such as were found were ascribed to narrowing of the spinal canal and compression of the cord. Clinical and radiological findings are fully described and before me these have not been questioned as a unique contribution to the clinical literature of what in other parts of the world is a rare complaint.

397. But supplementing these findings certain biochemical and pathological data (as to thirty of the forty-six cases studied only) are presented. In relation to these the authors state that they found 'significantly high levels of fluoride' in the blood, urine and bone ash of the patients studied (the last two would be expected) but no other biochemical or pathological abnormality. Table XIX reproduced from the article, but adapted to read ppm instead of mg per 100 ml states their findings.

398. Though the authors themselves claimed that these figures represent 'Significantly high levels of fluoride . . . compared with normal', these figures in fact would, if accurate, represent in some cases levels of fluoride so low as to be quite anomalous in relation to the findings of other workers.

399. The table should be studied. Some comments suggest themselves immediately:—

(*a*) No explanation is given why results of only thirty of the forty-six cases studied are given, nor for the data omitted particularly in blood, urine and bone ash content.

(*b*) No indication is given how the 'normal' figures are arrived at. In relation to the blood in particular, reported as 'trace', as we have seen the plasma maintains a constant level even in low fluoridation areas in normal subjects from 0.14 to 0.19 ppm. Whole blood values may be higher[3, 4]. It may be correct to describe this as a trace but in a matter calling for micro analysis it probably indicates a method of measurement lacking in sensitivity.

TABLE XIX

FLUORIDE CONCENTRATION IN SOIL, WATER, BLOOD, URINE AND BONE IN 23 CASES OF SKELETAL FLUOROSIS WITH AND WITHOUT NEUROLOGICAL COMPLICATIONS, REPORTED BY SINGH *et al.*

Case Number	Fluoride Content (ppm)				
	Soil	Water	Blood	Urine	Bone Ash
Normal	—	<1.0	Trace	<1.0	<300
Neurological—					
1	67	14.0	—	4.8	700
2		3.5	4.6	—	—
3		1.2	—	18.0	—
7	67	14.0	—	6.6	—
8	85	10.0	5.6	3.5	600
9		1.3	—	4.5	—
10		—	—	6.7	—
11		10.0	1.8	5.6	—
14		10.0	0.5	1.75	2,400
15		9.0	0.6	2.33	3,300
16		3.58	1.7	—	—
17		9.0	0.6	7.36	—
18		4.5	2.0	25.50	1,000
21		6.4	0.6	5.75	6,800
No neurological complications—					
22		6.4	1.3	10.4	3,500
23		3.5	—	—	—
24		2.2	—	—	—
25		2.2	1.1	—	—
26		1.38	0.8	8.3	—
27		1.82	3.2	5.7	—
28		1.67	1.8	4.2	—
29		1.88	1.1	3.2	—
30		2.70	1.2	4.2	—

Adapted from Singh (1961)[2] to read in ppm in lieu of mg per 100 ml.

(*c*) It is impossible to see any correlation or consistency in the figures given for each subject. Assuming a constant level of intake at the water levels stated, the urine figures do not bear any recognisable relation to them. The variations between the two are so striking that some explanation or comment is surely called for but none is given. It must be remembered that these are all cases of disabling skeletal fluorosis pointing to prolonged exposure to high levels of fluoride over a number of years. Nothing approaching a state of approximate balance between urinary rates and drinking water rates can be discerned.

(*d*) Referring to the figures given for bone ash, no ages are given, nor is an age given for the figure cited as the norm. It is stated however that of the forty-six patients investigated (the data for thirty only is presented) their ages ranged from 35 to 75, with an average of 51.6. The values found on analysis should be compared with the following found by other workers in relation to the same condition.

TABLE XX

Bone	Age	Fluoride in ash (ppm)	Normal bone fluoride content in persons of same age (ppm)[1]	Author
Vertebra	22	7,500	450 ± 225	Linsman[5]
Sternum		6,900		
Rib	37	20,830	690 ± 345	Kilborn[6]
Vertebra		18,650	670 ± 335	
Rib	48	15,900	850 ± 425	Wolf and Kerr[7]
Vertebra		19,880	830 ± 415	
Rib	52	9,900	910 ± 445	Roholm[8]
Vertebra		9,300	890 ± 455	
Rib	72	11,200	1,200 ± 600	
Vertebra		13,100	1,180 ± 590	

[1] Mean ± standard deviation.

Table compiled by the South African Commission (1966)[9] from the works of the authors cited.

400. It would seem that all of Singh's forty-six patients had positive clinical signs of skeletal fluorosis and all were confirmed radiologically. Twenty-one had compression paraplegia indicating a terminal state. This has been reported only from India[10]. Radiologically there was a generalised osteosclerosis classified as advanced in thirty cases (sclerosis complete and bones appeared structureless and chalky), as grade II in ten (trabeculae coalesced and appeared granular and amorphous) and the remainder as grade I (cancellous bones showed accentuation of trabecular pattern with a little increase in radiological density).

401. With these matters in mind Singh's bone ash fluoride levels should be compared with those found by others as shown in Table XXI which again I take from the Report of the South African Commission[16].

TABLE XXI

AVERAGE FLUORIDE CONCENTRATION IN BONE IN RELATION TO FLUORIDE INTAKE

Water or intake	*Fluoride in bone ash (ppm)*	*Bone changes*
Less than 1 ppm for 41 years	200-800	Nil
1.0 ppm for 50 years	600-2,200	Nil
2.6 ppm for 10-20 years	2,070-2,600	Nil
2.6 ppm for 41 years	2,810-3,400	Nil
4.0 ppm for 10-20 years	3,700-4,300	Nil
4.0 ppm for 48 years	4,490-5,420	Nil
4-8 ppm lifelong	Above 6,000	Few have non-disabling X-ray changes.
7.5 to 10 ppm lifelong	Above 6,000	Few have non-disabling osteosclerosis.
20-80 mg daily lifelong	13,000 or more	Disabling fluorosis.

Compiled from data of Zipkin *et al.*[12, 13], Linsman[5], Stevenson and Watson[14] and Leone *et al.*[15] and taken from the Report of the South African Commission[16].

402. Armstrong and Singer[11] in referring to Singh's figures for blood fluoride levels, suggest courteously (as befits a fellow author) but firmly that they are not to be relied on; indeed they suggest that on the basis of their experimental work with animals, some of the levels reported are incompatible with life. The learned authors of the South African Report reject them for reasons similar to those I have given above. But the matter does not stop there. The data given in Singh's table (Cases 3, 9, 24, 25, 26, 27, 28 and 29) has been used to advance the suggestion that, contrary to all European and American evidence[17, 15, 18] as Singh himself admits, disabling skeletal fluorosis may result from the prolonged ingestion of drinking water containing as low as 1.2 ppm of fluoride.

403. The argument also arises from the same paper that advanced dental fluorosis (mottling) may result at much lower levels of water fluoridation than those suggested by other authorities. I refer again to the paper. Fairly advanced dental fluorosis was present in thirty-eight patients. It was of grade II severity in eighteen (showing white opacities and distinct brown staining), while twenty showed grade III dental fluorosis (considerable pitting and staining). Though 25% of his advanced skeletal cases showed no mottling he states that in the field survey it was noticed that as much as 90% of the population had mottled teeth, though the degree is not stated.

404. To return to the table and the particular cases referred to. Assuming the accuracy of the water analysis cited, there is an immediate and obvious anomaly when the much higher urinary figures for the same cases are studied, i.e., they were excreting far more than they were taking in. They lead irresistably to the conclusion that either the figures are inaccurate (cf. his own statement, 'the drinking-water was uniformly high') or there was a high fluoride intake from other sources not taken into account.

405. The latter suggestion appears to be well supported. The very high summer temperatures of the region studied have already been referred to. Under the conditions described by Singh where the fluoride content of the water is high and the climate hot, the daily intake of fluoride from water has been estimated to reach 10 mg. or more, as against a normal intake from this

source of 1 mg. in a temperate climate. The following additional factors have been suggested as relevant:—

(*a*) The water ingested came from wells. There can be great seasonal variation in the fluoride content of well water depending on rainfall, evaporation and the amount of sediment which it may contain at particular times. Summer evaporation can concentrate the fluoride content[19] up to five times as has been noticed in bore waters in Australia[20]. Sediment may contain much more fluoride than the water in which it is suspended[21]. I quote from Largent[22]:

> 'The relative importance of particulate fluorides was missed in Algeria, Tunisia and Morocco for nearly ten years. So far as is known this aspect of exposures to fluorides in India (Shortt, 1937, and Singh, 1963) and China (Kilborn, 1950) has been largely ignored. At least there have been few or no balance studies to demonstrate how much more fluoride as a particulate substance is being ingested with food and water than is present solely as dissolved fluoride in liquids that are ingested there. In these areas the particulate fluorides may be of no importance as inhaled materials but the particles may add a very important amount of fluoride as a contaminant in the food eaten or dispersed (undissolved) in the water ingested. It seems likely to this author that fluoride, as a particulate, has played a much greater role than has fluoride dissolved in water amongst the cases of skeletal fluorosis reported in persons residing in Asia.'

The authors of the South African Report express themselves as being in complete agreement with these comments[23].

(*b*) Pandit *et al*[24] is regarded as establishing another source of high fluoride intake in that, in the population studied, local fluorite (calcium fluoride) and fluorapatite or phosphate rock was universally 'used for grinding foods and condiments'.

(*c*) A paper by Sita and Venkateswarlu[25] has directed attention to the importance of rock salt as a dietary item peculiar to the Punjab. They conclude:—

> 'If, in consideration of the extreme tropical climate of Punjab, we accept a daily consumption of 20 gm of NaCl as rock salt (200 ppm fluoride), the daily ingestion of fluoride could amount to as much as 4 mg. over and above that ingested through drinking water and other dietary sources, including tea, which is one of the important sources'.

(*d*) Apart from tea, Leatherman & Ellis[26] also mention other items of Indian diet, notably turmeric, a common ingredient of curry powder (29 to 32.5 ppm) beetroot and noolcole (70 to 140 ppm) as contributing to total intake.

(*e*) The poor nutritional status[27] of the subjects of Singh's study would probably have contributed to the fluorotic effects[19]. This has been borne out in other parts of the world but the exact relationship is uncertain.

406. None of these matters are sought to be evaluated in the studies referred to. In the absence of any such attempt the data (Table XIX) supplementing his clinical description of endemic fluorosis can hardly be said to be of any epidemiological significance in relation to fluoridation at 1 ppm in temperate zones and it does not constitute evidence giving rise to any reasonable apprehension of skeletal disorder in a Tasmanian community fluoridated at 1 ppm[3, 30, 4]. These studies were extensively and competently reviewed by the South African Commission but that part of the report[31] in which they are dealt with is too long to quote. However, the view I have stated accords with their conclusion.

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Section (d)—Kenhardt Disease

407. One puzzling study should be specifically mentioned. This is the matter of the pathological bony changes seen in native children as well as in adults in a reserve in the Kenhardt district in South Africa. They figure prominently in the writings of Professor Steyn, an opponent of fluoridation for whom I have much respect, and their photographs are produced in much of the antifluoridationist literature. They are perhaps most fully documented in a paper by Jackson[1] published in 'The Toxicology of Fluorine'.

408. I refer to the report as puzzling because, although the children concerned lived in a naturally fluoridated area with well water concentrations varying from 1.8 to 13.00 ppm (Jackson)[1] —16.1 to 3.4 ppm (Whittman *et al.*)[2] —and exhibited fluorotic mottling of the teeth, the bony symptoms were unusual even atypical of fluorosis, both in the fact that they occurred in children and also in their nature. This was in contrast to the normal classical osteosclerotic symptoms seen in adults in the same area. Dr Flynn, when giving evidence, in commenting on them as atypical expressed himself as convinced that the real cause of the disabilities was congenital syphilis. In doing so he relied principally on such clinical signs as tibial bowing (sabre

leg deformity), the notched teeth (Hutchinsons) shown in the photographs and the unsatisfactory nature of the evidence as to the means by which such a diagnosis had been excluded. In this regard he referred to the transcript of the cross-examination of Professor Steyn in the Irish High Court in the case of Ryan *v.* Attorney-General in which the same suggestion was advanced. He also referred to authorities which suggest that a diagnosis excluding syphilis could not be relied on in the absence of certain serological tests which it did not appear had been carried out.

409. The suggestion of syphilis, though superficially attractive, is speculative and can hardly be regarded as established. However, further inquiry is unnecessary. The South African Commission had an express investigation of this condition made by two members of the Medical Faculty of the University of Cape Town and their detailed and exhaustive report appears as Appendix J to the South African Report. Their conclusion, adopted by the South African Commission was[2]:

> 'Whatever the nature of this bone disease may be, it seemed to us improbable that excessive fluoride intake played a significant part in its aetiology or pathogenesis.'.

410. When investigated in 1967 the disease was disappearing, although the investigators could find no change in dietary habit or nutritional status to accord for any change in prevalence and despite a continued high fluoride intake (up to 16.1 ppm from one well). In view of the fact, as the authors' report, 'Despite a continued high fluoride intake and no obvious improvement in nutritional standards, the bone disease has virtually disappeared amongst children reared in the area'[2], it is difficult to see what, if any, force is to be attributed to the correlation previously claimed by Jackson, Steyn and others between its prevalence and fluorosis beyond that of coincidence. It has not in my opinion any implications adverse to the conclusions hitherto expressed.

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Section (e)—Skeletal Fluorosis—Conclusion

411. I think the matters discussed demonstrate clearly that with fluoridation at 1 ppm there is no reasonable apprehension of skeletal fluorosis of any kind. It is still true that the threshold level above which symptomless osteosclerosis may occur has not been definitely determined. It is certainly in excess of 4 ppm and probably not lower than 8 ppm[1]. What the level is in relation to disabling sclerosis, is more difficult to say. The South African Commission which made a specific study of the question have reported that in their opinion the:

> 'Dose likely to produce disabling skeletal fluorosis—approximately 20 mg per day for twenty years or more.
>
> 'Dose likely to produce radiological evidence of skeletal fluorosis without clinical disability—approximately 8 mg per day for twenty years or more[2].

412. I think these recommendations are justified by the evidence and indeed, under the prevailing climatic conditions of this State, I think they contain in themselves a substantial safety margin. Even if such levels of dosage are attained, it by no means follows that skeletal fluorosis of any kind will result. I have already remarked on the studies which show that even higher dosages are compatible with a complete absence of skeletal symptoms. In the Bartlett-Cameron study, only 10% of the group studied had detectable osteosclerosis and that was symptomless and, in fact, Largent[3] refers to a case of symptomless osteosclerosis in a workman exposed to industrial fluoride whose urinary output was 23 mg per litre. Below a fluoridation level of 6 ppm I could see no real risk of any normal individual even arriving at the threshold level of 8 mg per day for twenty years. The matter admits of monitoring. If check urinalysis of groups or individuals in respect of whom factors exist that lead to a suspicion of a higher level of exposure give levels less than 5 ppm, it can be safely assumed that no risk of harm exists[4].

413. I should add that no public authority, commission or other tribunal of inquiry over the last twenty years which has formally inquired into the matter, has reached a conclusion in any way opposed or substantially conflicting with what I have written. These include the Committees on Water Fluoridation set up in 1958 and 1967 by the World Health Organisation, the National Health and Medical Research Council of Australia, Commissions in New Zealand, South Africa and Ontario, the governing bodies of the American Medical Association and the Australian Medical Association, amongst others.

414. Professor Sir Arthur Amies[8] and Professor Steyn[5] did, it is true, refer to an adverse report of a select committee of the House of Representatives of the State of Michigan[6] which in 1964 recommended ' a two year moratorium on additional systems being ingested [*sic*] with fluoride until the subject could be further explored ' and some use of this Committee's Report has been made in other submissions. It seemed an unlikely authority but in case the Committee had brought something to light I asked for further information. Having obtained it[6, 7], I would suggest that the Committee and its Report belongs more properly to the turbulent eddies of American politics than to any realm of *bona fide* scientific inquiry. I consider it an unfortunate and ill-advised reference by the witnesses concerned who in other respects have been helpful. In any case the Report was not adopted and legislation introduced to give effect to it was rejected by the Legislature.

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PART H—THE ABNORMAL INDIVIDUAL

CHAPTER 23. THREE ALLEGED FATAL CASES

415. It remains to consider the case of the abnormal individual, and this is a matter to which the opponents of fluoridation have given a great deal of attention, quite properly. Much of the argument here is speculative but three reported fatal cases much relied on may be mentioned to give the matter some factual perspective. I should mention that Dr Waldbott[1] would refer to a fourth, that of a premature infant that survived birth for eighteen hours but the connection with fluoridation is, in my opinion, entirely suppositious and lacking in any kind of theoretical or practical confirmation.

416. The first case[2, 3] concerned a forty-one year old woman who had a long (twenty years or more) history of urinary disorder with periodic infection and hypertension (for eleven years). She also suffered from osteoporosis and severe osteomalacia. Except for a period from 1952 to 1957 her household water had less than 0.2 ppm of fluoride. From 1952 to 1957 it was fluoridated at 1 ppm; after 1957 it ceased to be fluoridated but she worked in a hospital which had fluoridated water where she had two cups of coffee a day. As she died in 1962 there would not seem to be any significance to be attached to her water history.

417. Following, in 1961, a complete breakdown of renal function after a virus infection (described as a terminal uremia) she was placed in an artificial kidney machine (a 'high flow apparatus') which used water fluoridated at 1 ppm. She survived eight months by repeated dialyses returning to her home in between. She died of congestive heart failure said to be primarily due to her hypertension. There were other symptoms of toxaemia. The use of fluoridated water in the artificial kidney machine resulted in the woman receiving 10 mg of fluoride in each four- to six-hourly period of dialysis. Since her kidneys and bone were unable to clear the fluoride there was a rise in the fluoride concentration in the serum. The authors suggest that this was to be expected but their discussion of its significance is inconclusive. The case may constitute a warning against the use of fluoridated water in artificial kidney machines. I am not qualified to say that it does but from this aspect it warrants consideration.

418. The second case reported by Linsman & McMurray[4] in 1942 was a case of a twenty-two year old soldier who died in a military hospital. He was a Texan. So far as I can make out from the report, his water history was:

Birth-7 years—Spur, Texas, 1.2 ppm.
7 years-9 years—Post, Texas, 5.7 ppm.
9 years-16 years—Lubbock, Texas, 4.4 ppm.
16 years-18 years—Washington D.C., no information.
18 years-21 years—Lubbock, Texas, 4.4 ppm.
21 years-22 years—Army Service, no information.

Temperature ranges for the places named are not stated. He had a history of injury to the right kidney at the age of fifteen, followed by hematuria (bleeding from the kidney) for one week and had had anaemic symptoms for some years previously. He died of acute renal failure. The pathological findings post mortem were many. They included extensive cystic inflammatory and infective changes in both kidneys, the ureters, the bladder and the prostate, sclerosis of bone (due to fluorosis) and dental fluorosis reported as severe in all teeth. In view of this and a similar condition being noted in a sister aged twenty, one questions the accuracy of the early water history. Bone fluoride content was above the critical levels previously discussed (Sternum 6,900 ppm ash lumbar vertebra 7,500 ash).

419. The authors of the report set out to describe the case as of interest because 'the condition of osteosclerosis in the bones of human beings who have incurred mottled enamel from this source (i.e. fluoride-containing drinking water) is not well known. As a matter of fact, no case of this kind has been reported in this country'. In their discussion they mention that the original clinical impression that the anaemia was secondary to the narrowing of the bone marrow spaces by the fluorosis was revised post mortem as the case showed 'abundant cellular marrow' and it was ascribed to the uremia. Such a condition as secondary to pronounced skeletal fluorosis is however not only feasible but well-known. They do not ascribe the kidney lesions to the fluorosis: they say:

> 'The chronic fluoremia may have aggravated existing kidney lesions. Or it is entirely possible that, because renal impairment was present, the osteosclerosis developed as a result of fluorine retention.'

420. I have set out the case at some length because of the importance attached to it by the opponents of fluoridation. It would seem however that it is no more than a case of acute and generalised renal failure associated with fluorotic osteosclerosis. No causal connection is claimed or demonstrated, though the possibility of terminal aggravation is admitted, but which one aggravated which is left open.

421. I pass now to the third case, again in the United States, reported by Sauerbrunn *et al.*[5] in 1965. This exhibited most of the features of the cases of skeletal fluorosis with neurological involvement previously only reported from India and which have been discussed in Chapter 22 (*c*) (*supra*). The patient was a sixty-four year old white Texan farmer. He was healthy before 'the late 40's' and was first admitted to hospital with skeletal and other symptoms in 1950. Subsequent admissions were in 1954, 1955, 1959 and 1961 and in 1962 when he died of terminal pneumonia. Besides the sclerosis with cord compression, the only other significant post mortem findings were dilation of bladder and ureters and inflammatory changes in the kidneys, but beyond suggesting that they could have resulted from functional urinary tract obstruction resulting from cord compression their significance was not otherwise evaluated.

422. His water history was obtained. He had been drinking for forty-three years water with concentrations of fluoride from 2.4 ppm to 3.5 ppm. But for twenty years before his first admission to hospital in 1950 he had been polyuric and polydipsic. The cause of this was not clearly established, though attributed by some physicians to diabetes insipidus. It had been clinically so considered at the time of his second admission. His family confirmed the polydipsia and that he 'always drank excessively large quantities of water and also drank a considerable amount of tea'. In 1950, when first admitted, his fluid intake and output of urine varied from four to ten litres per twenty-four hour period (seven to seventeen and a half pints). What it was at other times, e.g. when working, is not stated nor is any information given about temperatures. A sister who had lived with him for thirty-five years and a brother who worked with him and who had been exposed to 'identical fluoride concentration in their drinking water for the same period of time, had the same diet, lived under similar environmental circumstances' but differed only in the amount of water drunk, showed no symptoms of osteosclerosis but both had mottled teeth. The bone fluoride content was 6,100 ppm which agrees with the threshold levels previously discussed (Chapter 22 (a) (*supra*)).

423. The authors' summary of their conclusions is as follows:—

> 'This case of a patient with chronic fluoride intoxication, extensive osteosclerosis, and fluorotic radiculomyelopathy is believed to be the first reported from the United States. The development of advanced fluorosis in this patient exposed to drinking water with less than 4 ppm of fluoride was unusual and was probably a consequence of his excessive water intake.
>
> 'Prolonged polydipsia may be hazardous to persons who live in areas where the levels of fluoride in drinking water are not those usually associated with significant fluorosis.'

424. In this case the polydipsia had been present for thirty-two years before the patient's death—twenty years before his first admission. The suggestion advanced by Drs Exner and Waldbott that the polydipsia itself should be regarded as a symptom of fluoride toxicity is mainly based on the observation by Roholm that thirst may be a symptom of *acute* toxicity having been recorded in the symptomology of five out of thirty-four fatal cases that he surveyed[6]. But in his sixty-eight cryolite workers exhibiting signs of *chronic* fluorosis following a prolonged daily ingestion of fluoride which he estimated as between 0.2 to 1 mg per kg of body weight (*ibid* p. 201) excessive thirst was complained of by one woman only (*ibid* p. 137). All others questioned answered in the negative (*ibid* p. 301). While as a speculation it cannot perhaps be entirely dismissed, it was not considered by the reviewing physicians and has no confirmation elsewhere at the levels of exposure indicated.

425. As far as skeletal fluorosis is concerned, these three cases conform to what has been stated about the level of bone storage above which skeletal fluorosis may develop. While these remain the threshold levels, there are plenty of well attested cases in which those levels have been reached without symptoms of fluorosis[7, 8] and it should not be thought that such levels are themselves pathogenic[9]. How does one assess the significance of such reports? The authors claim novelty in the cases reported and it is impossible not to view them against the fact that as at 1965 there were 60 million people living in fluoridated areas in the United States, apart from the 3,000,000 or more who had for generations been living in naturally fluoridated areas, many at much higher concentrations than 1 ppm as well as hundreds of thousands in other parts of the world.

426. The further question, what causes such levels to produce symptoms in some and not in others cannot, it would seem, be definitely answered. Weatherell[9] considered it to be largely a question of the time and rate of absorption. 'If a bone level is achieved by a life-time's regular ingestion of small quantities of fluoride then no pathological changes will occur, whereas the same skeletal concentration, reached after a relatively short period, would be accompanied by symptoms of fluorosis'. But perhaps it is best considered as an open question. Skeletal storage of fluoride is a life-time process, though the rate decreases with age. Regular ingestion at a constant level leads to an approximate balance of ingestion and excretion, though a small proportion will always be retained. If the rate of ingestion is suddenly increased then during the time interval while the excretory organs are building up to a state of balance, there will be an increased retention percentage. Conversely, if the ingestion rate falls, a state of negative balance will be achieved and maintained for some time. (See Chapter 9 (*h*) (*supra*)).

427. The case of Sauerbrunn's patient (the only one in which the fluorosis had developed beyond an asymptomatic osteosclerosis) may also be considered from the point of view of total intake. The South African finding, which has ample support, was that the dose likely to produce disabling skeletal fluorosis was approximately 20 mg a day for twenty years or more. The subject's maximum daily intake of 10 litres of water if fluoridated at 3 ppm would have yielded 30 mg of fluorine. An average of 7 litres a day would still have given 21 mg. As against these figures should be set the estimate adopted by the National Health and Medical Research Council of Australia of the average daily intake of water of an adult in a temperate climate. This is 1.2 to 1.5 litres a day[10]. A comparable figure adopted by the Research Committee of the British Ministry of Health[11] has already been mentioned.

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CHAPTER 24. 'ALLERGY'

Section (a)—General Considerations

428. This is probably a good example of a concept which has outgrown its name. Medically the term is now recognised as inadequate and is perhaps in the discard but by the layman it is still used to indicate an abnormal physical reaction to a substance generally tolerated. In the lay sense it is a convenient heading under which to discuss a suggestion that in the community there are some individuals who, by reason either of constitutional idiosyncrasy or an acquired sensitivity may be unable to tolerate water fluoridated at 1 ppm.

429. No doubt there are some, and perhaps more than is generally realised, who, if led to fear that a substance is harmful, will react physically in the manner that their mental conditioning has led them to expect. Such psychosomatic symptoms may equally be demonstrated when, unbeknown to the sufferer, a neutral but indistinguishable substance is substituted for the original. The fact that such a class exists makes the discernment of a class of genuine sufferers difficult; it calls for the most careful scrutiny of diagnostic protocols and the rigid application of controlled experimental procedures.

430. In the case of fluoride, one is faced with the further difficulty that fluorides are so universally distributed in nature that a fluoride-free diet is impossible to achieve. It is a hallmark of most 'allergic' complaints that the amount of the offending substance needed to trigger off the distressing reaction is infinitesimally small. It is therefore difficult to see how one who claims to be allergic to a glass of water fluoridated at 1 ppm can yet depose to a life long habit of drinking ten to twelve cups of tea a day. If such an individual did exist the accidental drinking of some mouthfuls of sea-water would be fraught with danger. All of us have ingested fluoride incorporated in our bones and teeth and the process is continuous throughout life, including uterine life. The body maintains a plasma pool of the substance, which is continually being augmented from dietary sources and by reworking of the skeleton, while the kidneys concentrate and excrete any surplus to the body's requirements. The story of life is one of continuous exposure to fluoride as a ubiquitous element of the human environment. In physiological dosage it is difficult to conceive of it as a substance so foreign to the human body as to evoke the rejection response of an 'allergy'.

431. Originally the term 'allergy' was used to describe the rejection symptoms produced by a substance to which the body displayed an acquired and an excessive sensitivity. This sensitivity which in its effects could range from fatal and immediate collapse (anaphylaxis) to hives or hayfever, was due to the presence of antibodies in the system, resulting from prior dosage with the offending substances or allergens. It was not a normal reaction but an altered reaction due to induced sensitivity. The evidence is that in this sense, the substances which are capable of producing allergic reactions are invariably organic, either complex protein molecules, though carbohydrates and lipids may also be implicated, or increasingly the complex organic substances that are used in modern therapeutics. The inorganic fluoride ion falls into none of these categories, and the evidence is that in this sense an allergy to fluoride is neither theoretically demonstrable nor clinically recognised.

432. However, a further possibility exists in relation to an ill-defined class of substance known as haptens[1, 2], which, as I understand the matter, are not of themselves capable of acting as antigens but may do so when combined with a protein in the host[3]. The serum transport of fluoride is thought to be effected largely by a bio-chemical bonding with the serum albumin, not necessarily as a simple phenomenon, but more probably as a complex in which other ions play a part[4, 5]. If fluoride is, by reason of this (and there is no direct evidence that it is)[6], capable of producing the allergic effect claimed for it, then it would seem that this might be the type of mechanism which would be implicated[6]. However, in relation to fluoride I cannot see on the evidence that this can be ranked any higher than a theoretical speculation. It perhaps gains most support from the argument by analogy in that other halogens, iodine and bromine, have been implicated in this respect[2, 7]. However, the evidence of Dr Bristow, the consultant allergist called by the Commission to elucidate these difficulties, is that even in their case he has never known a reaction of the type here being considered to be produced by a dose of anything less than several thousand parts per million which would seem to indicate that such reactions in his experience may have been of the toxic variety only and not truly examples of allergy.

433. However, the variety of the human species is such that it is impossible to deny the possible existence of some individuals whose genetically determined characteristics include an abnormal response to any substance including fluoride. This is not a matter of an acquired sensitivity but of a constitutional defect making the individuals susceptible to toxic effects at levels greatly below those of normal species variation. By definition such a reaction should occur on the first exposure to the offending substance and it would have to be at a concentration exceeding the individual's prior experience and tolerance, whereas in the case of an allergic reaction the reaction is not experienced until multiple exposures have produced a state of sensitivity.

434. But this attempt to reduce the matter to two straightforward categories could be an oversimplification as true allergic subjects may as a class show a greater susceptibility to abnormal toxic reactions than ordinary people. This, as I understand it, is a matter of clinical observation, though the exact reason is unknown. But where in such cases, in the predisposing chain of causation, the true cause of the reaction lies, it is impossible to say. As I understand the evidence relating to such cases, even the most harmless of substances (i.e. to the population at large) can in combination with other substances and factors such as the current emotional, allergic and endocrine state of the individual concerned produce toxic symptoms. Whether that means that the final substance, the back breaking last straw as it were, in such a complex event is of itself noxious is not a matter that can be given an unequivocal answer. But I suggest these speculations in relation to fluoride are unimpressive unless based on accepted theory or clinically and specifically demonstrated.

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Section (b)—Complaints of Abnormal Reaction

435. Do such individuals exist? Attempts to demonstrate their existence have in most instances been singularly unconvincing but there is a residue of evidence which cannot be entirely dismissed. The case for the existence of such a group rests on:

(*a*) The work of Dr Spira who professed to recognise, as a result of some work during the Second World War, a great variety of symptoms attributable to fluoridated water (at that time naturally fluoridated of course) to which the name of 'The Spira Syndrome' has been given. The included such diverse matters as 'varicose veins and stammering, ambidextrousness, left-handedness, nail biting, defective vision, and dysmenorrhea, as well as feminism in men'.

(*b*) The submissions and the published works of Dr Waldbott who deposes to a number of cases investigated by himself in which toxic reactions to water fluoridated at 1 ppm were claimed as well as to other forms of fluoride administration.

(*c*) Some sporadic cases mentioned in the submissions made to the Commission which in the absence of any details of clinical investigations gain their maximum support from the names of the persons who introduced them.

(*d*) Other reported instances of adverse reaction to fluoride-containing dentifrices and solutions used in the painting of teeth. These include a reported study of abnormal reaction to fluoride tablets.

Section (c)—Dr Spira

436. Dr Spira (author of 'The Drama of Fluorine, Arch Enemy of Mankind')[1] can be quickly dismissed. At one time a prolific opponent of fluoridation, his work is now discounted, not only by the proponents of fluoridation but even by the more knowledgeable of its opponents, e.g. Sir Arthur Amies[2] and Professor Steyn[3]. Before me no-one of any consequence purported to rely on him, though he was cited by others. The thorough-going rejection of his theories on all sides, epitomised in Cox's remark[4]

'Spira's lack of understanding of the chemistry and physical behaviour of fluorine',

makes it unnecessary to consider the 'Spira syndrome' any further. His work received lengthy consideration as a prelude to dismissal in the New Zealand Report and to this and to others reference can be made if desired[3, 5].

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Section (d)—Dr Waldbott

437. The subject of Dr Waldbott is more difficult. He has made a submission for which I am grateful and I have been referred to many of his writings, both by himself and by the opponents of fluoridation to whom indeed he appears in the role of a leader. A controversial figure, both in himself and his work, in assessing both I am necessarily without the prime advantage of having seen and heard him and, of course, cross-examination and discussion has not been possible.

438. His academic qualifications are impressive. Graduating at Heidelberg, Germany in 1921, he has practised in America from 1923 to date. His speciality is allergy and he is a fellow and a former Vice President of the American College of Allergists. He is a fellow of other Institutes connected with his specialty, both national and international[1]. His work on allergy has received international recognition. He claims to have been the first to describe a number of important clinical entities including the role of pollen in chronic perennial asthma, allergic pneumonia, smokers' respiratory syndrome and anaphylaxis from penicillin[1]. While controversy surrounds these claims as well, his status and reputation as an allergist, at least prior to his espousal of the role of an antifluoridation advocate has been undoubted.

439. He first became involved in the fluoridation controversy in 1953 and has since been prominently identified with the opposition to fluoridation in common with his wife. Jointly, as I understand, they publish or are otherwise responsible for 'The Fluoridation News' which is a polemical journal. He is, as his supporters admit, emotional in his approach and frankly partisan. His later work tends to be an undiscriminating catalogue of references culled from world literature, the only criterion being it would seem that it can be tagged with an antifluoride label. In this, of course, he is not alone. It is a habit indulged in by both sides, but in the case of one who is so scornful of the scientific pretensions of those who disagree with him; who derides the qualifications as judges of the scientific literature, of bodies such as the Expert Committee of the World Health Organisation, it is a little disconcerting to find such an uncritical use made of so much of so little worth. This is particularly so when he goes outside the field of his own speciality, which is allergy; beyond that everything in the field of applied science is grist to his mill[2, 3, 4].

440. He believes that the 'U.S. Public Health Service had been bribed by the sugar and aluminium industry'[5] and that the scientists who support it have been duped or bribed by the carrot of research grants. Conversely, research into fluoridation has 'wrecked his career' and this belief is reflected in a bitterness in which it is difficult to discern any attempt at objectivity. His opponents are all either dishonest or incompetent or both and this ready resort to the argument *ad hominem* is one of the disturbing features of his later work. Editors who reject his work for publication are dishonestly motivated or in the grip of a conspiracy to silence all opponents of fluoridation. This is a claim repeated by others. The possibility that editors of reputable scientific journals with a world-wide reputation to maintain (and the more reputable the more frequent the accusation) may, even though mistakenly, honestly believe that the article is not worth printing because of a lack of intrinsic merit never seems to be entertained. He subscribes to the theory of a fluoridation conspiracy (he is reported by at least two sources as ascribing it to Communism[2]) designed to suppress the evidence of researchers and scientists and to promote the sale of fluorides as waste products of industry. This theme of a conspiracy runs throughout most of the more emotional anti-fluoridation literature. The evidence, or rather the arguments in favour of even its possibility, are so unconvincing that the beliefs of some of its most rabid supporters seem to verge on the delusional.

441. Attempts by others to investigate the cases of fluoride toxicity which he reports from his personal researches and clinical experience seem inevitably to end in bitter factual controversy which it is perhaps impossible to resolve[5, 6, 2, 7]. His opponents claim that he has, if not refused, at least hindered an impartial investigation of his claims. His reply is that the tribunals appointed were not impartial or that the data supplied has been misunderstood.

442. With this background, it is not surprising that his work is accepted by few and by none that I am aware of who have not espoused the same cause. But, as suggested by the late Mr Geo. Gray, who though a layman, has been very helpful to me in presenting the anti-fluoridation case by reason of his objective and reasonable approach, and echoed by Dr Sutton, fanaticism may conceal but not destroy his ability. As an allergist he is highly qualified. When, within the field of his speciality he says he conducted clinical examinations and tested his conclusions by recognised means, such as double blind trials and patch tests it is said his work should be evaluated on its own merit uncoloured by the emotional overtones with which other aspects of his work and

personality may be affected. In other words, the way it was put to me, he may be a fanatic and his emotional approach is regretted, but you cannot reject what he says he found unless you can say he is either incompetent or a liar.

443. Superficially this sounds reasonable, but to reduce a problem of evidence to those two alternatives as a problem in black and white is an over-simplification. Conscious subjective dishonesty is one thing, but the polarisation of evidence due to intense emotion is only too familiar, particularly in those fields in which, as in this, the subject matter can only be discerned by subjective interpretation of a minute and equivocal symptomatology.

444. Dr Waldbott[8, 9, 1, 10] claims personally to have investigated the cases of more than 100 patients exhibiting either a fluoride allergy or symptoms of fluoride intolerance. The latter he describes as his main interest[11]. In relation to both he has described a wide spectrum of symptoms affecting the neuromuscular, cardiorespiratory, gastrointestinal and urinary systems of the body as well as the skin. They range widely and the view has been expressed that in relation to allergy in particular they cover a symptomatology that is so diverse and lacking in specificity that it should be doubted on that account[12]. However, it appears that the author himself considers the allergic symptoms as part of the overall clinical picture of chronic fluoride intoxication, i.e., as I understand him, he is describing a concurrent and not necessarily an exclusive symptomatology. Not all his cases relate to fluoridated water. Some relate to dermatoses induced by the use of fluoride solutions, both in dentists and patients, as strong as 2% sodium fluoride or 8% stannous fluoride (a 1% solution is equivalent to 10,000 ppm) and others to toothpastes and welding fluxes.

445. These are not really relevant to the problem of drinking water fluoridated at 1 ppm. The subject of chemical dermatoses is too difficult to dispute the possibility of such occurrences. On the other hand, Menczer[2] drawing on experience of 100,000 applications of 2% stannous fluoride (four applications each to 25,000 school children), reports that not a single case of allergic reaction, either in the child or the dentist, or oral mucous response of any kind has been experienced. However, such dermatoses have been suspected, though rarely, by others and their possibility may be accepted. It is their relevancy which is unproven.

446. Dr Waldbott gives clinical but synoptic details as to how his diagnoses were established, covering such matters as double blind trials, patch tests and other customary weapons in the allergists' armoury. In some of them undoubtedly the diagnostic protocol leaves a great deal to be desired[13]. However in spite of those matters and in spite of the comprehensive symptomatology represented by the data described by him *prima facie* there would appear to be established in a few cases a reaction arising from fluoridated water which subsides on its withdrawal, and is excited by its subsequent administration.

447. The difficulty is the work has not been confirmed[14] and there is considerable doubt as to the diagnostic protocol used[13]. The latter is a matter of controversy. In the initial stages of his investigations there was a questionnaire used. Dr Waldbott says it was done to screen the patients with a view to subsequent investigation. The questionnaire and the answers were shown to a Dr Hornung, a German Officer of Health, who was investigating fluoridation in the United States and he has described it as leading, unscientific and generally inept. He gives examples taken from the first eighteen answers submitted and certainly to anybody but a believer supported by a boundless faith, it is hard to see the significance of such nonsensical material, even as a screening test[15]. Dr Waldbott[7] challenges, not the accuracy of the answers as recorded by Dr Hornung, but the allegation of their use in diagnosis, only.

448. However, one matter is said to be illuminating. One of the criticisms made of Dr Waldbott, prompted no doubt by Dr Hornung's revelations, was that he diagnosed some of the cases by correspondence. Such a procedure in the field of allergy, complicated as it is by subjective and psychosomatic difficulties, would I think throw his conclusions open to the severest doubt. When Dr Hornung's letter was produced to the Commission, I referred it to the late Mr Geo. Gray (President of the Anti-Fluoridation League of Australia) with a request that he obtain Dr Waldbott's comments. In Dr Waldbott's reply to Mr Gray[7] (produced to the Commission), there is the following paragraph:—

> 'This letter misrepresents the facts concerning my data on intoxication from drinking fluoridated water. Whereas I did use a questionnaire for the purpose of screening individuals who consulted me because of ill-effects from fluoridated water, I have never canvassed populations, as alleged by Dr Hornung; nor have I ever made a diagnosis of fluoride intoxication based on answers to a questionnaire.'.

This statement has been set alongside the following taken from another letter[16] by Dr Waldbott to the Editor of the Journal of the American Dental Association:—

> 'A questionnaire was used for preliminary orientation and for screening. In those not personally examined, laboratory data and examination findings were obtained from hospital records. A few cases were included in which the patient's history was so characteristic of the disease that it was, by itself, considered of adequate diagnostic significance.'.

I cannot at this distance pass a final opinion, but it does not serve to dispel doubts as to the diagnostic methods used.

449. I have not seen fit to discuss other factual controversies in which Dr Waldbott figures. At this distance it would be difficult to do so. His work has been critically reviewed on many occasions[13, 14]. Some of these criticisms are gathered and considered in an article 'Fluoridation and the Allergist' by Leonard F. Menczer[2] in which the conclusion expressed was that Waldbott's contention that the use of fluoride in caries prophylaxis 'will result in a wide range of physical manifestations is unsound, unscientific, and unprofessional'. Dr Bristow's opinion was that the comment was justified.

450. Dr Waldbott gave evidence in person before the Irish High Court in the Case of *Ryan* v. *Attorney-General for Ireland*[17] and before the Ontario Royal Commission, both of which made findings adverse to his contention. The latter tribunal discounted his method of presenting his data and found his evidence unconvincing[18]. The Research Committee of the British Ministry of Health has reported that in 1957[19] 'he submitted his evidence to the Councils on Foods and Nutrition and on Drugs of the American Medical Association, but failed to convince them that drinking water containing 1 ppm of fluoride was a hazard to health'. The report[20] of this body states in relation to Dr Waldbott:—

> 'Reports have been published or have otherwise been made available to the Councils of patients with symptoms and signs that were attributed to the ingestion of fluoridated water; to us, these reports fail to demonstrate enough consistency to justify impartial acceptance as showing a symptom complex due to fluoridated water.',

and this report was subsequently adopted by the House of Delegates of the American Medical Association[20]. The Expert Committee of the World Health Authority[21] in 1958 reported:—

> 'No confirmed cases of allergy to fluoride have been described in population groups drinking controlled or naturally fluoridated water.'.

In his editorial comment prefacing the World Health Monograph to be published later this year Professor Ericsson has said categorically:—

> 'Of the fears of harmful side-effects from controlled fluoride ingestion that have often been voiced, some can be immediately rejected on account of their obviously unrealistic nature; for example, this is the case with the allegation of allergy to fluoride, which is unknown among the world's billions of consumers of the fluorine-rich tea.'.

451. It is the lack of substantial confirmation, as well as the weight of expert opinion, that tells most seriously against the objective validity of the Waldbott observations. As the South African Commission said:—

> 'At first reading of his work there appears to be reasonable association between fluoride in the drinking water and allergic manifestations in the subjects he describes but the manner of presentation of his text [*sic*] protocols in his study leaves serious doubt as to the significance or validity of the work and in the absence of confirmation we regard his allegations as unsubstantiated.'.

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Section (e)—Other Allegations of Allergy or Toxic Response

452. The Commission received no satisfactory clinically verified evidence of allergy or abnormal toxic reaction to fluoride. I recall the following:—

(*a*) A lady emotionally fixed in opposition to fluoride and to doctors who claimed that notwithstanding a life-long habit of drinking twelve to fifteen cups of tea a day, she experienced dysentery and other pains some months or so after water fluoridation was introduced. She confirmed the self-diagnosis by drinking it again and exhibiting the same results. Since then she drinks unfluoridated water and is perfectly healthy. As the lady was quite well I did not consider it necessary to attempt to have this case medically investigated.

(*b*) A complaint (by letter) was received in respect of a Launceston family alleging a number of symptoms due to fluoride in water. As it had an intrinsic quality of reasonableness I considered that it should be investigated. A panel of leading physicians was submitted to the main sufferer and she chose Dr John Grove, a consultant physician well known in this State. His careful investigation reported at length leaves me in no doubt that the complaints were due to natural causes and in no way related to fluoride.

(*c*) I did receive a few complaints of subjective symptoms which though no doubt sincere, lacked any kind of clinical or objective verification. It is difficult to eliminate the psychosomatic element in communications of the nature that I have in mind. None were serious, all seemed to have made an adjustment which met current needs, and after considering them very carefully I did not think anything would be achieved by seeking to take them any further, with the exception of the case mentioned in the preceding paragraph.

(*d*) After the Commission had been in existence for nearly fifteen months, in fact on the second last day on which evidence was taken, four statutory declarations were produced from residents of various parts of New South Wales alleging physical disabilities due to fluoride. I blame no-one for their being so late but it has meant that no real opportunity was given to test them, as it was quite impracticable at that stage to contemplate further prolonging the Commission's proceedings while these matters were investigated in another State. I did, however, take the opportunity of discussing them in evidence with Dr Bristow, head of the Allergy Clinic of the Alfred Hospital. In his opinion only one of them might have been worth following up, the others he would be content to dismiss on their own histories. This one has been referred to the New South Wales authorities, as I knew they had followed up similar allegations in the past with, as far as I am aware from inquiries made, uniformly negative results.

(*e*) A letter was produced to the Commission which I accept as having been written by Dr William P. Murphy, a Nobel Prize Winner in Medicine for 1934, in which he claims to have a patient who is allergic to fluorides and who 'is unable to drink water so contaminated'. No details are given. Apart from the respect that one naturally accords to a physician of that attainment I have no means of evaluating the report.

(*f*) I was referred to a case of Mrs Gill, as it is reported at length in the British Medical Journal[1, 2]. I do not propose to discuss the controversy. Having read it I consider the alleged connection with fluoride unproven.

(*g*) I have ignored some other allegations which came to the Commission's notice as having no inherent semblance of trustworthiness. Generally speaking these were hearsay complaints, were obviously exaggerated and in some instances approached the ridiculous. They included one highly coloured account of people collapsing in the streets 'due to the fluoride in the water'. Unfortunately this turned out to be in a non-fluoridated town.

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Section (f)—Allergy or Intolerance to Dentifrices, Lotions, and Tablets

453. Allergic reactions, generally of the contact type, have been described as a result of industrial processes[1] in which fluorides have been used or liberated, particularly hydrogen fluoride. In the latter case it is the molecule as a whole and not the dissociated ion that is the toxic agent[2, 3]. Claims have also been made of similar reactions following the use of fluoridated toothpastes[4] and solutions used in the process of painting teeth as a caries preventive measure. Waldbott cites a number. In some cases no details are given of the strength of the fluoride application but I think it can be safely assumed that they represented concentrations some thousands of times higher than water containing 1 ppm. As reactions of this sort are known even with unfluoridated toothpastes, I do not think they have any particular relevance. However, I should say that the evidence is not undisputed. Menczer[5] records programmes involving approximately 100,000 topical applications of 2% NaF without 'a single case of mucous membrane irritation, stomatitis, aphthous ulcer or other allergic manifestation' and no such case has come to the notice of Dr Bristow as director of the Allergy Clinic of the Alfred Hospital, Melbourne. A solution of 2% NaF is close to neutrality. Modern alternatives such as 8% stannous fluoride and acidulated fluoride phosphate solution have a very low pH—that is they are very acid—and it is possible to get a transient reaction of the gums to their use. This is of no pathological or other significance to the matters discussed in this chapter.

454. In relation to fluoride tablets, many references were made to a study of Feltman and Kosel (1961)[6] which reported side effects in 1% of pregnant women and children up to eight years of age who had been given fluoride tablets. Unfortunately, the study having been designed to test the hypothesis that any beneficial effects from fluoride would be derived only while teeth were being formed and calcifying, the matter of side effects was an incidental issue only and was not reported, if it was investigated, with the particularity that would be warranted by the import- and later attached to it.

455. To assess the matter I have found it necessary to refer to all three reports[7, 8, 6] which relate to it and not merely to the last of them which was a common fault. The study was instituted in 1945. Tablets were given to expectant mothers during pregnancy and to the child after birth up to eight years of age. The first report mentions two types of tablet only, one of calcium fluoride and the other of sodium fluoride. Later, according to the second report, a third type of tablet was introduced of sodium monofluorophosphate (Na_2PO_3F) but when or why does not appear. All tablets yielded 1 mg of fluoride ion. Four groups were studied—

(i) Patients given tablets;

(ii) Controls from the same locality as (i) with no known supplemental fluoride;

(iii) Patients who drank artificially fluoridated water throughout their pregnancy; and

(iv) Controls from a nearby area that did not have a fluoridated water supply.

Tablets could be chewed, dissolved in the mouth or taken with any liquid according to the patient's preference. One tablet daily was prescribed, apparently for both children and adults and no distinction was made according to age. Each patient was specifically warned that the prescription was a poison if more than one was ingested daily. This could possess some significance.

456. In the second report (1956)[8], it is stated that of the 601 cases in the adult group, six showed undesirable side effects from fluoride tablets and five of the 495 children. This constitutes the 1% of the cases studied referred to in the final report. It is plain therefore that the final report includes no cases additional to those mentioned in the second report.

457. Of the adult cases, some detail is given as to four only. Three (two cases of vomiting and one of urticaria) were within two weeks of confinement and because of that 'it was not possible to investigate thoroughly the causes and relationship of the fluoride therapy and the symptoms'. In fact, it does not appear that any investigation at all was made, though the conclusion is expressed that:—

> 'At the time the cases were reported, it was concluded that these symptoms were not the result of fluoride ingestion, but a reaction to the binder used in the tablets.'[8].

In the absence of any investigation it is not clear even on what grounds this negative conclusion was reached.

458. Details are given of a fourth adult case in which the patient commenced taking the supplement in the fourth month of pregnancy; developed a rash after a week, but which cleared up on discontinuing the tablets. On resumption it reappeared in two days, this time accompanied by nausea. Tablets were withdrawn and symptoms disappeared. Resuming two weeks later the rash reappeared with vomiting and blood showing in the vomit. The symptoms disappeared on the tablets being withdrawn. The pregnancy was otherwise uneventful.

459. Of the children, details are given of two cases only. Both had urticarial symptoms (one with gastric symptoms as well) which disappeared when the tablets were withdrawn to reappear on subsequent administration. The tablets were discontinued with relief in both cases. No side effects were mentioned for any of the controls including those drinking fluoridated water.

460. In no case is the use of a placebo mentioned and on those details it is clear that one was not used in the only cases of which particulars are given. I think it is clear that if this was the extent of the investigations made they were inadequate to support a definite diagnosis of fluoride toxicity. In the circumstances I am unable on the evidence supplied by the authors in their own articles to understand the passage in the third and final report (1961)[6] which has been so much quoted:—

> 'One percent of our cases reacted adversely to the fluoride. By the use of placebos, it was definitely established that the fluoride and not the binder was the causative agent.'.

The statement could have reference to the two adults and four children concerning whom no details are given. But the position is hardly satisfactory. There are no other similar reports relating to tablets containing 1 mg of fluoride notwithstanding the free use being made of such tablets for similar purposes in many countries. It may be relevant to mention here that some of the dental aspects of this paper have been strongly criticised[9, 10].

461. However, minor gastric upset was noted in a few cases by Bernstein and Cohen[11] when administering large doses in capsule form (from 10 to 66 mg of sodium fluoride a day) in the treatment of osteoporosis and was 'frequently eliminated' by the use of enteric coated capsules, pointing to gastric irritation as a result of a concentrated dosage.

462. No doubt the Feltman and Kosel study points to a possibility of some form of abnormal reaction to tablets, though as against it Professor Martin gave evidence of an extensive programme of dietary supplementation by fluoride tablets conducted under the supervision of the Dental Department of the University of Sydney since 1945. Currently some 2,000 children are participating in the programme. In this study conducted over twenty-two years, there have been no reports of allergy to fluoride, either in tablet form or in solution, which have been substantiated. Three which did present with symptoms suggestive of fluoride toxicity presented the same symptoms when the fluoride was removed and a placebo substituted. A psychosomatic origin was demonstrated in each case. He also referred to another large scale programme of tablet administration to approximately 2,000 pregnant women conducted by the Medical and Dental Schools of Western Australia. Experience deposed to by Professor Storey in the administration of tablets by the Dental Faculty of the University of Melbourne paralleled that of the Sydney Faculty and has given no confirmation of the Feltman reaction.

463. Other reports[12] of reactions to fluoride tablets have lacked the clinical verification of adequate testing and elimination of psychosomatic factors.

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Section (g)—Medical Evidence

464. Because this is a difficult subject I sought information from any medical practitioners who might have had practical experience of fluoridation, both in this State and elsewhere, as well as the opinion of an Australian specialist in allergy. In respect of the former, my inquiries produced negative results, except one case of a complaint of constipation following the administration of 66 mg a day as a therapeutic measure to combat osteoporosis[1].

465. An inquiry made by a witness of the New South Wales Section of Allergy (Australian Medical Association N.S.W. Branch) produced the following reply[2] signed by the Secretary of the Section:—

> 'We have no personal experience of Allergy caused by Fluoridation in water but this may not be of any real significance on a statistical basis in New South Wales. We are aware, however, of some cases of either definite allergic troubles or strongly suspected allergic troubles from fluoridation from overseas records.
>
> 'We also feel that doubts cast upon the effectiveness of fluoridation, therapeutically, when taken in conjunction with many doubts about toxic and possible allergic reactions, has influenced us to state that we cannot feel that the use of fluoridation is without some risk, at least in the allergic field.'.

The principal reference of three given was to Dr Waldbott's book 'A Struggle with Titans'. The second was to a legislative document, the purpose of which I think the writer has misunderstood (see Part 'I', Chapter 37, *post*) while the third was to a review article of the type referred to by Dr Exner as a 'propaganda' article and which he suggests (and I agree with him) should be ignored. In addition a general statement was made indicating 'access to many other references from overseas', but the nature of those has not been elucidated[3].

466. Subsequent evidence[4, 5] and the references given made me suspect this was rather a naive and ill-considered answer. This suspicion was not removed by a subsequent communication[3] which *inter alia* indicated a misunderstanding as to how the letter came to be produced to the Commission. I merely record for information that the letter was not produced voluntarily by the person to whom it was addressed in breach of confidence as was implied in the letter referred to but under the coercion of cross-examination by counsel for the Pure Water Association who was apparently aware of its contents.

467. Because of these matters and because of the obvious difficulty of the subject, I sought the evidence of Dr Bristow, a leading Australian consultant in this field and the physician in charge of the allergy clinic at the Alfred Hospital in Melbourne. He had the advantage of having met Dr Waldbott, discussing fluoride with him and of having read his works.

468. Though water fluoridation is only in force in one small town in Victoria, the use of fluoride tablets, dentifrices and lotions for topical application has been a clinical commonplace for years. Not a single case of toxic or allergic reaction has come to his notice, either in his private practice, which is wholly confined to allergy, or in his clinical work at the Alfred Hospital. Nor has he become aware of any cases seen by other allergists and can recall none in the Australian literature. He is not, himself, satisfied of the objectivity of the reporting of Waldbott's cases and feels unable to accept his conclusions, particularly insofar as they seek to impute to fluoride the role of a sole cause in the symptomatology described. He does not, himself, consider that the fluoride ion is capable of acting as an allergan, but is prepared to accept the possibility that some people suffering from allergy in the true sense may further exhibit an intolerance to low levels of otherwise harmless substances, including fluoride. However, as to the possibility that such a response would be observed to water fluoridated at 1 ppm he said he could hardly imagine it happening. It has not been encountered in his practice or in that of other allergists with whom he is in contact and he suggested that many of the so-called cases of allergic or abnormal toxic response would on full investigation not be confirmed. If such did occur, and I understand he regarded it as no more than a speculative possibility, he would expect the symptoms produced to 'be merely a mild discomfort' such as a tendency to a rash or a minor gastro-intestinal upset.

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Section (h)—Conclusion

469. In relation to allergy and fluoride idiosyncrasy, the fundamental question is, do such individuals exist? I am by no means convinced that they do. But the categorical negative is unjustified. I think the residue of evidence in a doubtful field requires that one should accept the possibility that there could exist in the community some individuals who will react adversely to concentrations of fluoride at levels far below those which will affect normal beings. The evidence would indicate that any such abnormal reaction is far more likely to be a toxic idiosyncrasy rather than an allergy in the strict sense, hence its probability will increase as the dose. It follows the likelihood of any such reaction to water fluoridated at a level as low as 1 ppm is very remote, so remote that I cannot quantify it statistically.

470. Artificial fluoridation is now 25 years old, natural fluoridation immeasurably older. It is 15 years old in Tasmania, 7 or 8 years old in other parts of Australia. There are also naturally fluoridated waters in Australia, particularly in Queensland, though affecting small communities only. Tablets have been in use by thousands for years, many under clinically controlled programmes. The knowledge and experience of Dr Bristow cannot yield a case, nor is there one anywhere in the Australian scientific literature. In the light of the world status of fluoridation, these reported cases depending so largely on the testimony of one man, even if every one of them was established beyond controversy, would be of very doubtful significance.

CHAPTER 25. POLYDIPSIA AND POLYURIA

Section (a)—Diabetes

471. Another facet of the abnormal individual argument relates to individuals whose ingestion of fluoridated water or whose retention of fluoride may reach abnormal proportions because of disease. Diabetes and kidney disease are the two complaints around which the discussion has centred.

472. Diabetes can be quickly dismissed. There is no evidence that the prevalence or morbidity of diabetes is in any way affected by fluoride at 1 ppm, and no evidence can be demonstrated from vital statistics of any increased mortality[1, 2, 3, 4, 5, 6]. If, as a result of the excessive thirst that may accompany an uncontrolled diabetes, the fluid intake were increased to that of eight times the

normal man, the fluoride ingested would still only be equivalent to the intake of an inhabitant of Bartlett (8 ppm F) and studies in connection with that city have given negative results[6]. As the South African Commission concludes:—

> '. . . excessive thirst is a symptom of very poorly controlled diabetes, a state that, if allowed to persist, carries so serious a prognosis in its own right that the questionable hazard of increased fluoride ingestion assumes negligible proportions. We are entirely satisfied that the consumption of fluoridated water carries no special risk for the diabetic subject'[7].

473. In such a case it is the diabetes that should be treated, not the fluoridated water. A rather sensational statement was produced to the Commission to the effect that there were 100,000 'crypto-diabetics' in Australia, i.e., diabetics who are not aware of their condition. I have no means of checking the accuracy of this statement nor of knowing to what type of diabetes it was meant to refer. Such a crypto-diabetic would not be at risk unless as a result of polydipsia (excessive thirst) he exceeded normal fluid intake by the proportion mentioned. If after 10 to 20 years of polydipsia of the order mentioned he was still a crypto-diabetic, then no doubt he would be at risk of developing terminal skeletal symptoms. I refer again to the case reported by Sauerbrunn *et al.* (1965)[8] where the patient, a Texan farmer, a suspected diabetic, had been suffering from an uncontrolled polydipsia for 32 years before his death in an area where the water was fluoridated between 2.4 to 3.5 ppm.

474. I should mention that the South African Report at p. 29 concludes that in cases of polydipsia and polyuria, there is further safeguard against excessive retention in the ability of the kidney to concentrate fluoride and to excrete a higher percentage of the ingested dose as a result of the polyuria. The matter depends upon a technical discussion of kidney function which was not repeated before me. I think it sufficient merely to mention it here without comment. I have discussed it briefly in a previous chapter (Chapter 9 (h)).

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Section (b)—Kidney Disease

475. The role of the human kidney in urinary excretion makes it a subject of importance. More than any other organ in the body it is exposed to ingested fluoride and it has been extensively studied. Two questions recur:—

(*a*) Whether fluoride itself has a role in producing or aggravating a nephritic state; and

(*b*) Whether as a result of impaired or diseased kidneys a person drinking fluoridated water is subjected to the risk of fluoride retention with toxic effects.

476. As to the first, it should be unnecessary to observe that diseases of the kidney may be expected to occur in fluoridated areas as well as in non-fluoridated areas. A causative role for fluoridation at 1 ppm is not demonstrated merely because cases of nephritis occur in such areas. As in the case of diabetes, all studies based on comparative mortality or morbidity figures from fluoridated and non-fluoridated areas have entirely failed to show any significant association either way[1, 2, 3, 4, 5, 6]. H. C. Hodge[7], whose status as an expert on fluoride toxicity can hardly be questioned, takes the matter further. He makes the categorical statement that—

> 'Fluoride-induced renal pathology, with the causative agent established beyond reasonable doubt, has never been reported from chronic exposures in man. The nearest to such a finding came from the gross and histological studies of tissues of ten former cryolite workers by Roholm[18] who "stated as a fact that poisoning with cryolite produces *considerable changes of bones and ligaments, but no changes—or at the most doubtful changes—of the organs*".'*

*(Italics in Roholm's original text.)

Hodge then compares experimental renal pathology induced in animals exposed for prolonged periods to excessive amounts of fluoride and concludes that with the doubtful exception of one of Roholm's cases of a 'slight grade of chronic nephritis' in a patient exposed for 24 years to excessive quantities of cryolite dust no comparable human data can be found in the literature. To produce pathological changes in rats 100 ppm (roughly equivalent to a daily dose of 10 mg per kg) was found necessary for six months. Fifty ppm was insufficient and 100 ppm is regarded by Hodge as the 'borderline water concentration at which some individuals of certain species (but not all) exhibit characteristic changes'[7]. On the other hand, doses up to 320 mg of sodium fluoride a day for a period of five to six months were given to adult and elderly cancer patients and up to 200 mg daily to leukaemic children with no evidence of acute or chronic effects in the kidneys or in relation to any other vital function [8].

477. Hodge's review[7] is detailed but definite and entirely convincing and I accept it. The paper most relied on and commonly cited by opponents of fluoridation[9] is demolished by reference to another report from the same laboratory indicating a mistake by the authors. Incidently, another paper by the same authors was examined in detail by the New Zealand Commission and rejected as exhibiting 'unsatisfactory scientific features'[10]. The South African Commission made an independent examination of the first paper on its merits and concluded that it was open to 'serious criticism' for a failure 'to recognise as normal microphotographs of normal distal tubules' which they apparently thought were abnormal[11] on erroneous grounds.

478. As to the second matter, it would seem that, while renal function is maintained, renal clearance of fluoride at low concentrations is not markedly affected, if at all, by disease. This may be due to the physiological mechanism employed (See Chapter 9 (h) (*supra*)). Thus a study of two groups each of about a dozen elderly patients, one group with normal kidney function and the other with long standing kidney disease, was made of renal clearance, both before and after the fluoridation of drinking water began, without showing any significant difference[4] and chemically nephrectomised rabbits excreted the same amount of fluoride as control animals, even after ingesting it at 15 ppm for 42 days[4].

479. In cases of renal failure or seriously reduced function increased bone storage has been demonstrated[12, 13]. It is worth quoting from the last study referred to as it is cited by both sides:—

> 'The highest fluoride levels (i.e., of bone fluoride) were observed in older adults showing the end-stage kidney of bilateral pyelonephritis and polycystic disease. Average levels were found in subjects with unilateral pyelonephritis and in subjects with pyelonephritis with only slight to moderate disease. The highest fluoride levels found, in subjects with the most severe kidney disease, were within the normal range, and no disease associated with fluorides was evident'.

Skeletal retention can also be seen in the case reported by Linsman and McMurray[14] already noticed (Chapter 23, *supra*). However, while increased bone fluoride values may be expected at 1 ppm, there would appear to be no risk at all that even if all the fluoride ingested was retained that there would be any harmful effects[15,16].

480. Where bone breakdown, as well as complete renal failure, is present, then no homeostatic mechanisms to protect the serum level remain. This was apparently the case of the patient[17] exposed to the concentrated dosage of 10 mg in the artificial kidney machine but she survived long-term dialysis with fluoridated water for eight months. This is the only case of this nature reported.

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CHAPTER 26. VARIATIONS IN DIETARY HABIT

481. The fluoride intake resulting from the average American diet, apart from drinking water, has been estimated at 0.25 to 0.3 mg daily[1]. This has been tested by urinalysis conducted in widely scattered areas in the United States where the drinking water contained only traces of fluoride, so that urinary fluoride could be attributed wholly to and used as a measure of food ingested fluoride[2]. McClure also mentions that similar data have been obtained in Oslo, Norway. A similar figure was adopted by the National Health and Medical Research Council as applicable to Australian communities[3]. Taking tea into account Hodge (1963)[4] would put the dietary contribution at 1.3 to 1.8 mg per day. Longwell (1957)[5] whose work is generally cited calculated the probable intake from all sources for men, women and children in Great Britain as shown in Tables XXII and XXIII. In so doing he used the results of a pilot study of the liquid consumption from all sources of 207 people living in a London suburb.

TABLE XXII

CONSUMPTION OF LIQUIDS IN GREAT BRITAIN

	Average cc per day						
	Water	*Milk*	*Beer or Cider*	*Tea*	*Coffee, Cocoa*	*Soft Drinks*	*Total*
Men	100	414	230	1,100	71	29	1,944
Women	86	386	29	843	57	14	1,415
Children 5-14 years	200	443	....	286	14	86	1,029

Longwell (1957)[5] citing the results of a pilot study by Dr R. E. Bransby of the Ministry of Health.

TABLE XXIII

AVERAGE INTAKE OF FLUORIDE MG F PER DAY

	Water containing no fluoride	*Fluoridated Water 1 ppm F*
Men	1.8	3.2
Women	1.3	2.2
Children 5-14 years	0.6	1.2

Taken from Longwell (1957)[5].

These figures were adopted by the Research Committee set up by the British Ministry of Health and other Governmental authorities in 1962[6]. As 1,000 cc equals 1 litre it will be seen these are generous estimates. They provide, I would think, a conservative basis on which to consider safety factors connected with diet.

482. It is argued that there are some members of the community who, as a result of dietary habit or working conditions, may have a much higher daily intake of fluid. This is no doubt true, though exact figures are difficult to obtain. Nikiforuk and Fraser[7] in reviewing the literature as to children concluded that the individual variation in consumption within any given age group amounts to as much as a seven fold difference between the lowest and highest rates of total tap water consumption. Marier and Rose[8] in a limited sample found a three fold variation (from 1,020 ml to 3,150 ml a day) among seven members of a research staff over a Canadian winter month. Under conditions of extreme industrial temperatures, e.g. bakehouses and furnace rooms, the adult variation would no doubt be greater. Dr Polya suggested a possible figure of thirty pints or seventeen litres (taken I gather from Bicknell[9]) a day and this was accepted by Dr Flynn. Somebody else, I think, without citing authority, suggested nearly as many gallons! Dr Exner[10] claimed a recorded case (in 1856) with a daily water consumption of eight to twelve gallons, though it seems this may have been due to diabetes, and that a daily consumption of fifty to sixty pints is not uncommon. With U.S. pints this would be six gallons (Imperial) a day, but such levels were beyond the experience of all other witnesses even under the conditions of heavy industry. Assuming they may be met with, they are still well below the level at which any acute symptoms of fluoride toxicity could be expected.

483. The difficulty again is to arrive at the relation between ingestion and absorption. Working conditions which produce an excessive demand for fluids will also be likely to induce sweating. The sweat glands are important organs in fluoride excretion and under conditions of severe sweating the fluoride loss can be extremely high, from 13% to nearly 50% of the intake[11, 12, 13]. Under conditions of induced sweating Crosby and Shepherd (1957)[12, 14] have shown an increase in the total excretion (i.e. urine plus sweat) and if this be the case then the net percentage gain would be smaller than in normal subjects. This was accepted by the South African Commission[15] which also accepts the fact that increased urinary volume will have the same effect. Age is also a factor in retention. While the amount retained by the skeleton increases with age the rate of retention diminishes[16, 17].

484. Any danger lies in persistent intake. Intermittent high intakes do not constitute such a problem, as the process of building up a physiological balance between the amount retained and the level of intake is a prolonged one and the process is reversible[18, 19], that is to say, if the intake drops, deposited fluoride will be mobilised and excreted until a new physiological balance is attained. I am unable to see any problems arising from the occasional 'bender'; however an intake of seventeen litres a day, if persisted in for twenty years, would put the subject at risk, but the margin is large and it is not just a matter of mathematics.

485. Two special dietary factors which have been frequently mentioned are tea and fish. I am not satisfied that fish is important even when eaten in significant quantities. While it is high in fluoride content, the fluoride is in the form of apatite and is doubtfully available. The only study which has suggested a toxic intake (to the extent of dental fluorosis) from a large fish diet (Sognnaes *et al.* in respect of Tristan da Cunha[20, 21]) is now questioned[22]. (See Chapter 21 (*e*) (*supra*)). Minoguchi[23] reviews a number of dietary studies and reaches the conclusion that the Japanese diet would probably contain 1 mg to 2 mg a day more than the American diets studied but makes it quite clear that this in no way represents the amount absorbed which would be considerably less[23].

486. Tea, on the other hand, is a different matter. It is unique in its capacity to concentrate fluoride and on infusion much of it will be released in ionic form and will therefore be assimilable. For this reason it has been of much interest to British, New Zealand, Australian, South African and Japanese researchers. In its dried leaf form it may range in fluoride content as high as 400 ppm but the Indian and Ceylon brands are much lower than the Chinese. The amount extracted in the infusion will be proportional to the amount of tea used and time spent in brewing and whether the pot is 'topped up'. However there is substantial agreement that using unfluoridated water the strength of the average infusion of medium strength would be about 1.0 ppm[24, 25] though Harrison (1949)[26] in the most detailed study of all would indicate about 0.75 ppm as an average for a normal brew. These figures obviously reflect traditional English methods of preparation; McClure using 'tea bags' got much lower figures, viz. 0.1 ppm[1].

487. Different writers use different measures. By test the contents of a large breakfast cup would yield about six fluid ozs, leaving out the fraction by which the level of the filled cup falls below the rim and the amount that may be left in the cup after drinking. Six such cups to the litre would seem to be a reasonable standard of computation and is the one used by Jackson and Weidmann[25].

488. There is substantial agreement that apart from the content of the water used, one litre of tea a day (six large cups) would yield 1 mg of dietary fluoride, though Harrison (*supra*) again indicates that this is slightly on the high side. There is some evidence that not all the fluoride in tea is absorbed which may depend on whether it is drunk with meals or not[26, 27]. If the water is itself fluoridated then the yield will be approximately doubled. Taking all these factors into account I think a reasonable estimate, which has some margin on the side of safety, would be to assume that seven average cups of tea a day made with fluoridated water could yield up to 2 mg of fluoride.

489. This figure is based on an approximate tea consumption of 1 litre. Using a slightly higher figure (1,100 cc) Longwell[5] has calculated that the total intake of fluoride from all dietary sources for the English male would be equal to 3.2 mg a day. This calculation was adopted by the Research Committee of the Ministry of Health in its 1962 Report[6]. If the tea intake were doubled the yield could be up to 5.2 mg a day, but it would still be below toxic levels.

490. I quote and adopt the following statement taken from the Report of the Research Committee of the British Ministry of Health[6] at p. 47:

> 'Tea is the principal source of fluoride in the usual diet in this country. A freshly prepared infusion contains about 1 ppm and the fluoride intake of heavy tea drinkers may, therefore, be above the average level of the areas where they live. It has been estimated, however, that a person living in an area where the fluoride content of the water was 1 ppm would have to consume roughly a gallon of tea (30 cups) daily to reach a fluoride intake approaching the average of the residents of Bartlett, U.S.A. Moreover, this calculation, which assumes that the average fluid consumption at Bartlett is the same as in Great Britain, is probably an underestimate because Bartlett is situated on latitude 30° North and therefore has a warmer climate than that of Great Britain. It is thus reasonable to believe that the fluoride intake of even the heaviest tea drinkers in areas of Great Britain with water fluoridated at 1 ppm would be unlikely to equal the average intake of residents of Bartlett, where no harmful effects could be detected.'

491. The results of the Bartlett study have already been given. They were non-disabling and symptomless osteosclerosis in 15% of those examined at ages from 25 to 80 years.

492. The matter of fluoridated water in tea drinking communities was specifically adverted to in a study by Jackson and Weidmann[25] of the bone fluoride content of deceased residents of three different English communities, viz. Leeds (virtually no fluoride) South Shields (0.8 at the time of the paper but previously 1.2 ppm) and West Hartlepool (1.9 ppm). Comparisons were made with other figures obtained from London residents and from residents of Rochester in the United States of America, both non-fluoride areas, and generally with the results of the Bartlett-Cameron study. In general, bone specimens from the English non-fluoride areas had higher bone fluoride values than comparable specimens from the United States and this was ascribed to the difference in dietary intake represented by the consumption of tea. In respect of South Shields (0.8 to 1.2 ppm) the authors concluded that the fluoride values were characteristic of those in a tea drinking community whose drinking water has been artificially fluoridated. After reviewing the data, they stated:

> 'It can be concluded, therefore, that the artificial fluoridation of drinking water to the extent of 1 ppm does not enable bone to accumulate fluorine to the level that is considered toxic. This conclusion, however, applies only to circumstances in a temperate zone. In hotter climates there is a greater fluid intake and hence, for a given fluorine concentration in the drinking water, a relatively greater intake of fluorine leading to a greater accumulation in the bone'[25].

The results from West Hartlepool (1.9 ppm) were regarded as undesirably high but still as being below the level at which skeletal fluorosis was to be expected, though a few of the higher values were just above the lower limit of the critical threshold or index for bone as estimated by these authors[25].

493. Specific studies have been made of the tea drinking habits of Australian children in Queensland[28] and in Western Australia[14]. Some data has also been collected in Tasmania in respect of both primary school children and high school children. This was obtained from dietary survey records prepared by the Nutrition Officer of the Department of Public Health and related to twenty-eight separate surveys made over nine years in respect of fifteen separate primary schools and four high schools in different parts of the State. The average amount of tea drunk in the case of primary schools was 1.2 cups per day per child and 1.5 cups for the high schools. The range in both cases was 0 to 7 cups. These results were not, generally speaking, dissimilar to those obtained in Western Australia and Queensland (although hotter climates) where the same high intake was experienced in respect of a relatively few individuals. A regular consumption of seven cups of tea made with fluoridated water a day could yield up to 2 mg of fluoride a day. Add other sources of dietary intake and one could predicate for those particular children a total maximum intake of 2.2 to 2.3 mg a day. This is high, not so high as to constitute any danger to health, but high enough to entail the risk of dental fluorosis for a child under 8 years of age persistently drinking this amount of tea every day.

494. It is interesting to compare this estimated maximum intake of 2.2 to 2.3 mg with the results obtained by Kruger[28] in his survey of the fluid intake of Queensland children under 8 years of age. He gave specific attention to the problem of the few children that his survey disclosed as having a high tea intake with an overall high fluid consumption. Six children (one in Longreach, two in Nambour and three in Innisfail) were found in this category. Assuming an optimum amount of fluoride to have been added to the water supply (adjusted by Galagan's formula to the prevailing temperatures) he calculated a hypothetical daily intake on the part of those children of 2.72 mg, 2.14 mg, 2.2 mg, 3.05 mg, 3.17 mg and 2.03 mg. As he points out, these would be figures of ingestion only. Sweat and urine may reduce the amount retained, sweat by as much as 50%[12]. Following the matter through, the risk of dental fluorosis in children exhibiting this high dietary intake may be estimated by taking the following figures prepared in respect of communities in which the water has been naturally fluoridated at values higher than 1 ppm. Table XXIV[29] gives estimates of the amount of fluoride ingested from food and water by children under normal conditions in the United States when the water supply contains 1 ppm. Assuming the water concentration to be raised to 2 ppm the results for 7 to 9 year olds would then read as shown in Table XXV which is reasonably close to our postulated figure of 2.2 to 2.3 mg.

495. Galesburg (F 1.9 ppm) and Elmhurst (F 1.8 ppm) may be taken as possible parallels. Table XXVI shows the fluorosis values found by Dean[33] for those two communities and they may be used as bases for our theoretical comparison. The results would indicate that children under eight who habitually drink seven large cups of tea a day would have a 1 to 2% chance of 'moderate' dental fluorosis with brown staining, and a 6 to 8% chance of 'mild' fluorosis without such stains.

496. I have chosen tea as the only dietary item to be discussed at length because it is, in my opinion, the most significant, though many others were mentioned. In relation to tea I have no exact evidence of the tea drinking habits of the community. I have assumed, as have all witnesses, that our habits in this regard conform closely to those of British and New Zealand communities (though not to some Eastern countries where green tea with a much higher fluoride content is drunk). As a check, however, I have confirmed from official statistics that tea imports have not fallen off beween 1935 and 1965, in fact per head of population there has been a very slight increase.

497. Beer has been suggested as a dietary item approaching tea in importance. This was not confirmed by the evidence. However, the fluoride content of a number of Australian beers was obtained. In six cases out of eight they gave fluoride trace values only (less than 0.05 ppm) and the other two were 0.15 and 0.18 ppm[30]. Another beer was identified which I was given to understand was made with fluoridated water and a number of test analyses were obtained. Somewhat surprisingly the analyses uniformly indicated that the finished product contained less fluoride (little more than half) than the water which had been used in the brewing, i.e. some had been lost in the manufacturing process. Having confirmed the fact I was not concerned to inquire the reason but I have since learnt that this has also been recorded overseas[31]. I would conclude that the excessive beer drinker is less at risk except perhaps in relation to his pocket and his liver than the excessive tea drinker and this I think has been the conclusion of all those who have investigated the matter. The associated suggestion that fluoridation may interfere with brewing processes is baseless. There are large and famous breweries that have been operating in naturally fluoridated areas, e.g. Milwaukee and Wisconsin, for many years without difficulty or problems. Indeed there is some evidence that it is beneficial to brewing[31, 32].

TABLE XXIV

SUMMARY OF ESTIMATED DAILY INTAKE OF FLUORINE FROM FOOD AND FROM DRINKING WATER

(Drinking Water containing 1 Part per Million of Fluorine and Dry Substance of Food containing 0.1 to 1 Part per Million of Fluorine)

Age, Years	*Body Weight** kg	*Daily Fluorine Intake*			
		From Drinking Water mg	*From Food* mg	*Total Daily from Food and Drinking Water* mg	*Total Daily mg/kg of Body Weight*
1 to 3	8 to 16	0.390-0.560	0.027-0.265	0.417-0.825	0.026-0.103
4 to 6	13 to 24	0.520-0.745	0.036-0.360	0.556-1.105	0.023-0.085
7 to 9	16 to 35	0.650-0.930	0.045-0.450	0.695-1.380	0.020-0.068
10 to 12 ..	25 to 54	0.810-1.165	0.056-0.560	0.866-1.725	0.016-0.069

* Figures for weight for ages 1 to 6 years were taken from tables arranged by Woodbury (1921): figures for 6 to 12 years were taken from the Baldwin-Wood weight-height-age table for boys and girls of school age, published by the American Child Health Association.

Taken from McClure (1943)[29].

TABLE XXV

From Drinking Water	*From Food*	*Total Daily*
1.300-1.860	0.045-0.450 mg	1.345-2.210

TABLE XXVI

INCIDENCE AND DISTRIBUTION OF ENDEMIC DENTAL FLUOROSIS (MOTTLED ENAMEL) IN 7257 SELECTED WHITE SCHOOL CHILDREN, AGED 12-14 YEARS, CLASSIFIED ACCORDING TO THE DEGREE OF AFFECTION

City	F in ppm	No.	Distribution of signs of dental fluorosis						Community index* of fluorosis
			Absent		Present				
			Normal	Questionable	White opaque		Brown stain		
					Very mild	Mild	Moderate	Severe	
Galesburg ..	1.9	273	69	74	110	17	3	0	0.69
Elmhurst ..	1.8	170	48	54	51	15	2	0	0.67
			Percent						% prevalence
Galesburg ..	1.9	100	25.3	27.1	40.3	6.2	1.1	0.0	47.6
Elmhurst ..	1.8	100	28.2	31.8	30.0	8.8	1.2	0.0	40.0

*Public health significance of index: 0.0-0.4, negative; 0.4-0.6, border line; 0.6-1.0, slight; 1.0-2.0, medium; 2.0-3.0, marked; 3.0-4.0, very marked.

Extract taken from Table XI of Dean (1946) [33].

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CHAPTER 27. HEIGHTENED INTAKE DUE TO INDUSTRIAL EXPOSURE

498. Fluorides are a component of many industrial wastes. They are also found in high concentrations in some raw materials, notably in the cryolite (54% approximately) used as an electrolyte in the aluminium industry. They are found in lesser strength (3 to 4%) in the phosphate rock used in the fertiliser industry but relative quantities used are higher. In such industries, workmen handling those materials and exposed to the dust and fumes in retort and other mill areas will have a heightened intake of fluoride due to their working conditions[1, 2]. Such industries may also contribute to atmospheric pollution by the discharge of fluoride-bearing smokes and gases. This may pose a problem of local contamination not confined to the workers of the particular industry but affecting the inhabitants of the neighbourhood generally.

499. I am conscious that standards of health and safety, as they may relate to industrial processes in which fluorides are used or emitted, are not within my terms of reference. Such standards, I am informed, do exist in other parts of the world but not apparently as a matter of legislation in Tasmania. That is not to say there is no awareness of such matters.

500. Fluorides are freely absorbed from the respiratory tract either as inhaled gas or as phosphate rock dust[3]. My concept of the abnormal individual is wide enough to include those who, by reason of direct industrial exposure or atmospheric pollution, may take into their systems such quantities of fluoride as to make it undesirable that they should receive any supplement from drinking water. If such a class is to be found in any given community, then, in the absence of other remedy, it may constitute a valid and cogent argument against the fluoridation of communal water supplies to which they, in common with others, must have recourse.

501. However, in relation to atmospheric pollution some perspective must be retained. This is a matter of degree. I have already referred to the fact that fluorides are a normal constituent of the environment and their presence in the atmosphere, even in areas far removed from industry[4], is normal and inevitable. In 1961 it was estimated that some 25,000 tons of fluorides were discharged into the atmosphere annually in England and Wales[5] but such total figures are really meaningless as they give not the slightest indication of local concentrations. Volumetric measurements taken in the centre of London indicated a normal winter pollution of 0.1 to 0.15 micrograms of total fluorides, gaseous and particulate, per cubic metre of air. In contrast, measurements of total fluorides over the winter months at Rotherham and Stoke-on-Trent yielded an average of 0.7 and 1.9 micrograms respectively. Values in air samples recorded by Call *et al.*[6] in industrial areas in Utah where the atmospheric fluoride was known to be elevated, ranged from 0.10 to 0.80 micrograms with an average yearly value of 0.24 micrograms per cubic metre.

502. In 1949 an elaborate investigation was conducted by the Fluorosis Committee of the British Medical Research Council at Fort William in Scotland to assess the health hazard due to the establishment of an aluminum industry in the neighbourhood. The investigation and the report[7] which resulted from it is of great interest, not only because of its detail, but also because at the time of the investigation the fumes emitted from the furnace rooms were not scrubbed or treated in any way but were allowed to escape 'in a dense white cloud'. By contrast efficient 'scrubbing' with over 90% fluoride extraction is now claimed by both the major Tasmanian industries which are concerned with this problem, though in the case of the aluminium industry at Bell Bay, this position was not attained until 1967 owing to technical problems. This makes the figures of dispersal obtained at Fort William of great interest. Concentrations of fluoride in the furnace rooms ranged from 0.14 to 3.43 mg per cubic metre of air; the higher values being obtained from an old furnace room without extractor fans and blacked out under war-time conditions. Most of the other samples would have been below 1 mg per cubic metre. It was found by test of the actual smoke cloud that 'The total fluorine content of the air decreases rapidly until at 200 yards it is about fifteen per cent of the mean concentration in the furnace rooms, and is then approximately halved after a further 240 yards; and it is halved again after another half mile. At one mile the concentration had fallen to three per cent of the mean for the furnace rooms.'.

503. I have no series of atmospheric readings for Tasmania. A test reading taken in the concluding stages of the evidence at a point ¼ mile from the E.Z. Works at Risdon in the line of the prevailing wind yielded a value of less than 1.4 micrograms per cubic metre, but, as a single reading, its value is of course limited.

504. However, taking the winter readings for Rotherham and Stoke-on-Trent said by Martin[5] to be among 'the most heavily polluted areas of the country' of 0.7 and 1.9 micrograms per cubic metre as representative of a highly industrial area, then it can be calculated that the average man

engaged on moderately strenuous work and inhaling 20 cubic metres of air a day, would ingest from atmospheric sources but 0.014 and 0.038 milligrams of fluoride a day which is not significant. This is allowing 100% absorption of atmospheric fluoride whether gaseous or particulate[8]. These calculations explain the comment of Largent[9]:—

> 'Generally, there have been no effects on the health of man from the inhalation of airborne inorganic fluorides within his place of residence. Any effects of any kind induced by airborne fluorides have concerned (1) the few instances reported where fluoride was present in the atmosphere within the area where men were at work or (2) where the fluoride became a contaminant which was ingested with food or water or both. Dusts have not been released in communities adjacent to industrial operations in amounts sufficient to influence the intake of fluoride by man.',

which reflects the general view. Perhaps the last sentence should be qualified to the extent that in the study by Call *et al.*[6] already referred to, higher concentrations than average were found in the bones of elderly people with advanced kidney disease (pointing to increase in bone storage as balancing a decreased urinary excretion rate) in areas of high industrial pollution. But 'the highest fluoride levels found, in subjects with the most severe kidney disease, were within the normal range, and no disease associated with fluorides was evident'. This conforms with Roholm's experience who found no abnormalities attributable to fluoride in the population of areas of great industrial pollution[10].

505. There are, as I have indicated, two major Tasmanian industries in respect of whom it has been suggested these matters are relevant. In regard to the E. Z. Co. at Risdon, I have some evidence of the discharge of 250 lb of fluoride a day into the atmosphere. I have no information as to the composition or dispersal of these products except the isolated reading of less than 1.4 micrograms already referred to. The company concerned quote a computed value of ground level gas contamination of 1.25 mg per cubic metre which they say 'is a theoretical ultimate maximum, which would arise only under unusual weather conditions and then only at a point in fairly close proximity to the base of the stack'. Particulates are not referred to though in close proximity to the stack these would be the more important[5]. However, I think it is possible they were meant to be included and I will so assume. But this figure is really useless as far as indicating atmospheric pollution in the environs of the factory and of doubtful value even as indicating conditions within the mill as can be appreciated from the qualifications attached to it. If it were otherwise it would be a high figure as can be seen from the Fort William study.

506. In relation to this computed figure of 1.25 mg the company have referred to a threshold limit value of 2.5 mg per cubic metre recommended by the Occupational Health Committee of the National Health and Medical Research Council of Australia of 1964[11] which has been adopted from an American source[12] but this is a time-weighted average concentration for a normal workday of eight hours[2], i.e., it is an index fixed by reference to a working period of, I assume, forty hours a week and is not an index of community air pollution or air pollution nuisance or of the toxic potential of continuous uninterrupted exposure[11, 13].

507. However, from the aspect of environmental pollution, air measurements are not perhaps the best measure of exposure. Urinalysis and a careful watch for dental fluorosis in children would be a much better indication of excess. While I have no present reason for concluding that there is any likelihood of air pollution significantly contributing to the fluoride intake of inhabitants of the area surrounding the Zinc Works, in view of the paucity of the information and in view of the circumstances discovered in relation to Bell Bay I would recommend as a matter of public health reassurance that some effort be made to survey children living in the immediate neighbourhood before any extension of fluoridation is considered in the area.

508. As to the Aluminium Works at Bell Bay, I have evidence which makes me suspect that there has been an environmental problem in relation to the George Town area. Again I have no air figures. Some figures representing air analyses of furnace room atmosphere taken some years ago gave concentrations expressed in milligrams which were, if not lethal, so close to the lethal range that they were obviously inaccurate and it so appeared, in that milligrams had been substituted for micrograms. The company has since corrected them. As corrected they are within the range of the lower Fort William values but the company's extraction methods would now appear to be much more efficient. The company maintains and has done for some years, an environmental check in relation to plant contamination but this is not regarded as a reliable indication of human environmental problems[5]. Significantly, however, the results do point to a recent increase in pollution in the George Town area itself.

509. A dental survey made for the purposes of the Commission in January 1968 of children in the George Town area has disclosed a degree of dental fluorosis which makes it probable that they and the inhabitants of the area as a whole (and not merely those exposed to dust or fumes by reason of working in the plant) are receiving an amount of fluoride exceeding in degree anything that can be related to normal dietary sources or to their water supply which is not fluoridated. The excess is not great and it raises no serious problems but the level of dental fluorosis, of mild and very mild classification, is significantly higher than it is in nearby Beaconsfield which is fluoridated. The low level of caries experience of the children examined is seen as confirmation.

510. The probable source is air pollution, though the matter requires further investigation. However, the probability is strong enough to make me endorse without hesitation the recommendation of Dr Brothers, the Departmental Consultant, that fluoridation of the water supply serving the George Town township should not be considered until the situation has been more closely evaluated. A factor which will have to be taken into account is the improvement that may be expected from the operation of the fume towers and scrubbing plant which have only become fully operative since the latter part of 1967. Before that date the evidence is that they were only approximately 20% operative.

511. I have considered whether the matters discussed in relation to George Town have implications concerning Beaconsfield. The evidence is they do not and I accept it. Three factors have influenced me:—

(*a*) The lower fluoride levels obtained for plant samples in the area.

(*b*) The evidence as to the degree of dental fluorosis observed in Beaconsfield, which is within normal values and below that in George Town, even though Beaconsfield is fluoridated.

(*c*) The greater distance, 5 miles as against 3, of the two townships from the plant and the difference in the prevailing wind.

However as Beauty Point is served by the same water supply as Beaconsfield the matter should be watched. It is another matter indicating the need for surveillance of fluoridation projects at an expert level of public health.

512. There remains the problem of the effect of direct industrial exposure. Since 1937 when Roholm established industrial fluorosis as a pathological entity, clinically manifest fluorosis has been rarely reported. This is generally considered to be due to the awareness produced by Roholm's report, to improved industrial conditions and to higher extraction and recovery rates of expressed fluorides. The Fort William investigation found only minimal effects—' Some of the older workers, who had been exposed for many years to concentrations probably higher than those generally prevailing at the time of the investigation, showed X-ray changes in bone which could be ascribed to fluorosis; in none of these workers, however, was there any clinical disability '[14]. This despite full exposure of up to fifteen years and twenty-four-hour urinary samples giving values as high as 23 mg. Similar findings have been recorded in a valuable study of the phosphate fertiliser industry by Derryberry *et al.* (1963) but the threshold conditions described underscore the need for vigilance[2]. Other relevant references are Collings (1952)[3], Brailsford (1953)[15], Martin (1967)[5] and Hodge and Smith[16].

513. I have no evidence of any clinically manifest industrial fluorosis in Tasmania. It would be easy to detect incipient fluorosis if it exists by urinalysis of those with a history of prolonged exposure to high concentrations followed by X-ray examination. However, no such evidence in relation to Tasmanian workers who may, by reason of prolonged industrial exposure, be assumed to be at risk is available. As the matter does not fall directly within my terms of reference I have not sought it other than in the terms of the general inquiries mentioned in this chapter. The Aluminium Works at George Town have taken some spot urinary samples in connection with biennial medical examinations which they carry out under sect. 19 of the Workers' (Occupational Diseases) Relief Fund Act 1954—though fluorosis is not a ' disease ' to which the Act is directed—and have supplied me with the results. These have disclosed some high urinary concentrations. In 1961 and 1962 the figures showed 7% of the staff tested had urinary values of 10 mg per litre (i.e., 10 ppm) or above while 54% in all were above 4 mg. Tests made in 1967 of forty-four employees chosen as a cross section exemplifying both degree and length of exposure, showed such diverse results that I would hesitate to draw any conclusions from them except possibly as to the unsatisfactory nature of spot samples[8]. However, one sample exceeded 10 mg per litre while in all 32% had values of 4 mg per litre or higher (up to 9.2 mg). I have no values for the E.Z. Co. at Risdon and do not know whether similar examinations have been carried out. It could be that they have but the value for fluorides not taken.

514. My problem, however, is not industrial fluorosis *per se* but the implications of adding fluoride to the water supply and thus adding perhaps up to 4 mg a day to the intake of any individuals living in the community who may by virtue of industrial conditions be already at risk.

515. In the case of the Aluminium Works I am clear there are some who would be put at risk by water fluoridation if they are not already, which I think probable. It is impossible to be complacent about urinary outputs of 10 mg per litre if they were to be confirmed as indicating a constant rate of exposure of long standing[17, 18]. As to the E.Z. Coy., I have no information and I express no opinion either way. Their computed value for gaseous concentration at ground level makes it possible. At 1.2 mg per cubic metre of air, 6 to 7 mg could be inhaled per eight-hour shift, but the computed figure is high in relation to other figures given in the Fort William Survey and by Comalco, where nevertheless higher individual intakes are suggested by the urinary excretion rates.

516. A daily intake of 20 mg a day for ten to twenty years is regarded by Hodge[19] as the danger level for crippling fluorosis, but to forestall osteosclerosis (which may be without disability) he considers, citing D. A. Irwin[20], that the optimum intake should not exceed 5 mg a day. This is not a mathematical level and probably under 8 mg a day the risk of actual disability is nil, though bony changes may be seen[17, 18]. Above it, after lengthy and continuous exposure, I would consider the individual at risk. Moreover, it must be stressed that these figures relate to total absorption from both occupational and non-occupational sources and not merely to one factor.

517. It would seem, therefore, that the industries mentioned pose a problem, though I do not know its extent, i.e., what percentage of the total work force is involved, or its degree. In all probability it would be small. As to Comalco, 90% of the work force live, I am informed, in George Town but some cross the river and live at Beauty Point and Beaconsfield which is a fluoridated area. The percentage may increase, of course, when the Batman Bridge comes into use. Meanwhile my recommendation as to George Town would operate to protect any workers, residents of the township who may be potential subjects of industrial fluorosis. As far as the Zinc Works is concerned, the problem is suspected but not confirmed, and I have no means of estimating its extent or the dispersal of its workers among the populations of the cities of Hobart and Glenorchy.

518. The view taken by the South African Commission[21] was that the answer to such a problem is industrial and that the remedy should be sought at the main source of exposure. The risk that may exist in such cases would not exist primarily as a result of water fluoridation but as a result of industrial conditions. If as a result of such conditions the intake of the few men exposed is brought to the level where water fluoridation constitutes in their case a hazard, then their exposure is probably high enough to constitute a hazard in any case and measures to control it should be sought as a matter of industrial safeguard.

519. I have mentioned only two industries. Fluoride may be released as dust or fumes in other industries, notably any in which there is a heavy consumption of coal, and in steel making, brick and tile making and the chemical industry generally[22, 5]. There may be other problems of industrial safety involved in those industries where organic compounds such as Teflon are used[23] though those are not directly relevant to water fluoridation. A State or community which introduces water fluoridation cannot neglect to examine standards of industrial safety in relation thereto. Specifically in relation to fluoride, air pollution does not seem to be a major problem, but the George Town experience which, in the light of recorded experiences in the literature of the subject, seems to me anomalous, should be borne in mind.

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CHAPTER 28. THE ABNORMAL INDIVIDUAL, CONCLUSION

520. I have taken some pains with this topic because I would agree that it is a hallmark of a civilized society that the rights of minorities, even of individuals, are of importance. To the normal individual fluoridation at 1 ppm constitutes no danger, brings no disadvantages. To the young its advantages are manifest. Even to the toothless adult, more probably than not, it confers a benefit. But the case of the abnormal individual has seemed to me to be of importance. If there are such individuals, and if the possibility of harm is substantial and substantiated this must obviously be a consideration that would give pause. But, if the community advantages be undoubted, then such a risk to be countervailing must be more than theoretical and the existence of the individual who would be affected must be more than a speculation. There must be some identifiable element of the population to whom these considerations can be applied. And ultimately, there must be a fair balance struck between all relevant factors—between the advantages and the risks.

521. I have set out what I believe to be the relevant factors. There is a risk of 'mild' dental fluorosis in approximately 2% of children. It will not be disfiguring or even ordinarily noticeable except that children who for some time preceding their eighth birthday have had an abnormal fluid intake, leading them to consume seven large cups of tea a day, would, if the tea were made with fluoridated water, have approximately a 2% chance of 'moderate' mottling or brown staining. While this mottling is of fluorotic origin, it is to be expected that there will at least be a corresponding reduction of mottling and staining due to other causes. It may be even greater. The risk

of endemic skeletal fluorosis is I believe nil except possibly under uncontrolled industrial conditions in relation to workers in some industries. The risk of some individual achieving such a state by reason of disease induced thirst, kidney breakdown or excessive dietary habit is so remote that I cannot quantify it. The only cases cited bear little relation to fluoridation of water at 1 ppm.

522. Lastly, in relation to the abnormal individual, I have sought to examine the cases of the so-called allergic or abnormally intolerant individuals. I am by no means sure that they do exist, though I admit the possibility. If they do exist, the statistical significance of such individuals is so small, the consequent harm so slight, and once recognised so easily prevented, that in relation to the expected benefits of fluoride I can see no real balance being achieved.

PART I—MATTERS, MAINLY MEDICAL, RAISED BY WAY OF OBJECTION TO FLUORIDATION

CHAPTER 29. GENERAL

Section (a)—Some Absurdities

524. The opponents of fluoridation may well ask to be preserved from their friends. There are responsible men among them, men of undoubted attainments such as Professor Steyn of South Africa and Sir Arthur Amies and Dr Sutton of Australia and among the laymen the late Mr Geo. Gray (M.H.R.) and Mr Davenport to name but a few. On the other hand there are some who in the fixity and strength of their delusions, approach the paranoidal. Under this head of objections the initial task has been to distinguish between the substantial and the trivial, the rational and the reverse.

525. I was informed that the 'official and professional campaigns in favour of fluoridation are the most dangerous and subversive propaganda yet to appear in the Western World[1]. That it, fluoridation, was responsible for the First World War and the Russian Revolution. That it was a Nazi plot to achieve world domination, but that when the Russians invaded Poland 'the German and Russian general staffs exchanged scientific military plans, the scheme of mass control through water medication fitted into the Russian Communist plan to communise the world '[2]. This is not the isolated statement of some deluded visionary, but is to be found repeated over and over again in the voluminous literature of the subject with which the public intelligence has been assaulted over the years. I quote Exhibits 76[3], 168[2], 213[4] and 768[5] as examples only. In another printed pamphlet[6] portions of which were allegedly produced in evidence to the Public Health Committee of an American State Legislature, the use of fluoridation as a focal planning point in a war of aggression is developed over several pages of strategic nonsense. To others, however, it is a Zionist plot or the nefarious design of some vague group of international financiers, but the idea is the same—to dominate the world.

526. Very often these and other allied statements are ascribed to persons with impressive medical, scientific and academic attainments. The title 'American Association of Medico-Physical Research' is impressive, almost a guarantee of authority, until one learns that among the accomplishments of its founder is the ability to diagnose any disease from one drop of the patient's blood and that he could by his 'electronic reaction' machine cure any illness[7, 8]. A B.A. degree is not necessarily an indication of scientific knowledge and is not always a sufficient guarantee that the writer's views are not a farrago of chemical nonsense[9]. One of the oddest in this category is a statement ascribed to the Rev. L. F. Sheen of the United States. There is, of course, a well-known Roman Catholic bishop and publicist of a similar name and the person who produced the statement admitted that he thought this much respected prelate was responsible. It has been reproduced in many other publications[10] seemingly under the same impression. It is a chemically ignorant statement which confuses the element fluorine with the ion of the element and adds for good measure some nonsense about fluoridated water smelling 'so foul' because it is decomposed. I thought this was an unlikely source for such a silly and crude statement and had it investigated. It was not, I learnt, the statement of the person to whom it had mistakenly been attributed, but of some obscure though related cleric with no particular competence, according to his bishop, in the field of fluoridation and who was speaking not for the church of which he was a member but as a private citizen[11].

527. Some of these statements obviously belong to the quasi criminal side of medical quackery[12]. Others are just as plainly misguided, though no doubt honest.

528. This is a sample[2]:—

> 'This is how it works—in the rear occiput of the left lobe of the brain there is a small area of brain tissue that is responsible for the individual's power to resist domination. Infinitesimal doses of Fluorine will in time *gradually* REDUCE the individual's power to resist domination by slowly poisoning and narcoticising this area of brain tissue and make him submissive to the will of those who wish to govern him. When the Nazis decided to go into Poland, the German and Russian general staffs exchanged scientific military plans—the scheme of mass control through water medication fitted into the Russian Communist Plan to communise the world. In Perkins' own words I quote " I was told of this whole scheme by a German Chemist who was an official of the Great Farben Chemical Industries and also a prominent Official in the Nazis movement at the time. The German's statement was 'I say this with all earnestness and sincerity of a scientist who

spent 20 years in chemistry, biochemistry, physiology and pathology . . . any person who drinks artificially fluoridated water for a period of 12 months or more will never again be that same person *Physically or mentally.*' " '.

This is a verbatim extract from a document produced to the Commission by a medical opponent of fluoridation.

529. Accordingly, fluoridation is claimed to resemble a 'chemical lobotomy' producing 'a lackadaisical attitude with over-indulgence in SEX, ALCOHOL, sleep, food, and spending money'. Also that it is responsible for an increase in 'gross indecency and child molestation cases'[4, 6]. It is therefore not surprising that it has been blamed for an increase in juvenile delinquency[12], for the failure of elementary school children to pass examinations[6] and that it is 'undermining Christian standards by the forces of darkness'[13] elsewhere referred to as the 'Organised Powers of Evil'.

530. Another allegation constantly repeated for which there is certainly no vestige of proof, in relation to the Australian scene, is that fluoridation is a promotional device of the aluminium industry for the disposal of an otherwise unsaleable by-product. In fact the evidence is that in so far as it is economic to seek to recover waste fluorides they are readily re-usable within the industry that produced them.

531. Normally, one would ignore such excesses as well as statements that in a Tasmanian town fluoridated water was causing people to fall about in the streets (this report, incidentally, related to a non-fluoridated town), that it caused the tea to go black (I could not ascertain whether this was with or without milk), that it cracked dentures and radiators and killed flowers. Equally the confusion that exists in the cruder forms of opponents' literature between fluorine the element and its ion, between organic and inorganic compounds of the element as illustrated by the *in terrorem* hodge-podge of references to rat poison, nerve gases, Compound 1080, etc., all expressed in pseudo scientific jargon and with specious semanticism, would hardly call for comment were it not for its prevalence and for its obvious intention of appalling the ignorant. Probably at least a hundred of the papers, pamphlets, brochures and other documents produced to me fall in this class. They are rubbish and should be ignored by both opponents and proponents alike. Unfortunately, because of the distribution methods employed, lay members of the public are very susceptible to their influences. They should be disregarded. The case against fluoridation does not depend on this nonsense masquerading as science, even if it has an M.D. or a D.D.S. or a Ph.D. at the top (there are examples of all). It is, I am sure, an embarrassment to the more responsible of the opponents of fluoridation, but it does suggest very strongly the difficulty accurately of informing the public mind in the face of such a flood of suggestive misinformation.

532. I have mentioned this type of material in order to inform of its existence and its prevalence. I do not intend to discuss it further. There are responsible opponents of fluoridation and there are serious matters to be discussed. The kind of literature just discussed obscures rather than reveals the real merits of the controversy.

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Section (b)—That the Safety of Artificial Fluoridation has been Insufficiently Tested

533. Whatever may be urged by way of theoretical possibilities or speculative analogy, common sense will always find it difficult to discount actual experience. The answer of profluoridationists that the safety of fluoridation is attested by the fact that some 70 million Americans, as well as countless thousands in other parts of the world (not to mention at least a hundred thousand Australians), are drinking fluoridated water without apparent harm, is not wholly met by the rejoinder that clinical experience and conclusions based thereon may be, and sometimes have been wrong. Such a rejoinder seeks to ignore all matters of degree, the size of the populations concerned, the time over which observations extend and the skill, responsibility and integrity of those who might make the observations. All these are dogmatically swept aside in favour of the axiomatic postulate that clinical beliefs without scientific proof (whatever that may mean in the ultimate) are not to be relied upon. I am not really concerned to debate the matter but I would have thought it at least possible to argue the contrary case that in those areas where mistakes have been made, such as the much quoted one of thalidomide, it was clinical observation and acumen that incriminated the offending substance.

534. Some opponents seek to achieve an air of reasonableness by suggesting a time limit over which the trials should continue before general fluoridation is adopted. In the early literature 20 years was suggested. That has now been exceeded and there is a suggestion that 40 may be needed. To the answer that some millions have been drinking naturally fluoridated water in America, Great Britain and elsewhere for generations without, at the levels recommended for fluoridation, any known catastrophic or even inconvenient results[1], several answers are made. One is that there is a difference between naturally fluoridated waters and those in which it is added as a matter of artificial supplement. As Professor Polya, himself an opponent of fluoridation, said at one stage, this argument partakes of the mystical. It certainly has no chemical basis. It tended in some to be linked with theories of nutrition which, for lack of a better term I call naturistic and to be accompanied by a readiness to accept and swallow large quantities of vitamins and other 'Vitalistic' substances purchased usually by mail order from 'unethical' (in the pharmaceutical sense) sources.

535. Others less gullible have replied by way of objection that there may be qualitative differences between the mineral components of naturally fluoridated waters and those to which fluoride is added as a matter of artificial supplement. This could, of course, be completely met by the use of calcium fluoride as the additive and I presume for this reason it has been attempted in some cities. But it, calcium fluoride, does create engineering problems because of its relative insolubility and for that reason only it is not recommended. This objection I will later deal with more fully in a chapter in which the general subject of hard and soft drinking water is discussed (Ch. 41).

536. Still others, and these are the ones whose objections must from their nature be considered seriously, have replied that there are sufficient reported cases of harm resulting from fluoridated water to raise a presumption that its continued use may be unsafe. While in relation to the population exposed such reports are indeed meagre, I will endeavour to survey them to the best of my ability and to indicate how far they should serve to inhibit fluoridation proposals, but with one qualification.

537. The list of ailments for which fluoridation has, in the course of this dispute, been blamed at one time or another, is so extensive as to beggar description. From dandruff to tinea, acne to abortion, cancer to haemophilia, blindness to bedwetting, sterility to stammering, most known ills to which the flesh is heir, have been laid at its door. I do not attempt to deal comprehensively with such complaints, but only with those which in the light of the evidence given before me I have thought to possess some importance or interest. Detailed refutations of many more will be found in the highly expert South African report[2], in the New Zealand Report[3], the Ontario Report[4] and in the publication 'Classification and Appraisal of Objections to Fluoridation'[5]. An intemperate attack on this latter publication by Dr Exner[6] has been drawn to my attention but is belied in its substance by the information given by the authors in the document itself. Insofar as it relates to matters not urged before me and not specifically covered by other sections of this Report, it provides a useful reference point for those who would inquire further and I would recommend it as such.

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Section (c)—Professor Steyn and the South African Report

538. In this context the South African Report in particular assumes considerable importance. Professor D. G. Steyn is one of the opponents of fluoridation whose scientific attainments command respect. He shows considerable mastery of the literature but unlike others would seem at all times to have refused to step down from the courteous moderation of scientific debate to the crude polemics indulged in by some who, perhaps with similar attainments, have espoused the same cause. He is the more impressive on that account.

539. At the outset of its inquiry the South African Commission, the membership of which included five highly qualified scientists, requested Professor Steyn with Professor Staz (the latter a supporter of fluoridation and the author of a previous Official Report on the subject)[1] to attend all public meetings of the Commission so that they could assist in the questioning of witnesses. This was done. In this way the Commission obtained the direct assistance of one of the most expert of the opponents of fluoridation in evaluating what scientific bases there are for objecting to it, in particular, in relation to those matters of objection, mainly concerned with the thyroid gland and with the action of cortico-steroids which Professor Steyn has personally advocated. Their discussion of Professor Steyn's evidence is detailed and illuminating, but it is obvious that before this jury of his scientific peers, while they acknowledge with gratitude the help he gave them, he failed completely to substantiate theses general and particular, by which he sought to support his objections. I rely on this Report to supplement my own in relation to many medical aspects of the fluoridation controversy on which my report is silent.

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Section (d)—The 'Onus of Proof' Argument

540. A constantly recurring argument found expression in legal terms that the onus of proof (i.e., of safety) was on those who promoted fluoridation. This no doubt is true. But if it be correct to appeal to evidentiary maxims, their limitations should be recognised. For the legal doctrine to which such frequent recourse was made does not postulate an absolute unvarying burden, irrespective of the evidence adduced, without some qualification. Lawyers are accustomed to recognise an ambiguity in the phrase and to distinguish between a persuasive burden, i.e., an onus of final persuasion on all the evidence, and an evidentiary burden, i.e., of adducing evidence either by way of establishing an initial case or of rebutting a case *prima facie* established by the other side. An onus of the first sort does not change, but the second does, and may change frequently in a given matter according to the quantity and quality of the evidence adduced by one side or the other. This is not a matter of abstruse legal theory, but a proposition which on analysis is no more than practical application of a principal applied by all reasonable men in any process of judgment. It is particularly applicable when the matter to be proved is in essence a negative, and the propostion that fluoridation can do no harm is such a negative.

541. Absolute proof of such a negative proposition is an impossibility. To report as did in 1962 the Research Committee appointed by the United Kingdom Ministry of Health[1] that—

> 'Every general practitioner in each of the three fluoridation study areas was asked at intervals throughout the five year period during which the dental examinations were performed for information as to any harm arising out of fluoridation . . . From 89 doctors repeatedly questioned in the three study areas, only one reply was received which called for investigation . . . Investigations . . . into this possibility showed that the incidence . . . was not related to the water supply.
>
> 'Had any individual been thought by his doctor to be suffering from ill-health as a result of fluoridation it would have been desirable, provided that he and his doctor consented to investigate his condition more closely. It had therefore been arranged that the services of a consulting physician would be available followed, if necessary, by hospital accommodation under the National Health Service. But the appointed consultant still awaits his first case',

still does not prove absolutely that such a case may not occur in the next week or in the next year or in the next 50 years, but common sense would suggest it goes a long way and that the stage may

have been reached when those resisting the proposals might well be asked what evidence have they got to rebut the inference, persuasive though less than absolute, of safety as established on the evidence so far adduced.

542. This is my summation of this argument. I regret using legal conceptual terms, but this argument was so frequently advanced without any real appreciation of its limitations that I have been left with no option. The great preponderance of the evidence favours the complete safety of fluoridation, except in relation to those matters I have discussed under the head of fluorosis and a small qualification I make under the head of allergy. To continue to rely on the catch cry of the onus of proof without advancing some reasonably cogent evidence which would justify a continuing doubt is not to advance the matter.

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Section (e)—That Safety Should be 'Guaranteed'

543. Another variant of the same argument is the claim that safety should be guaranteed. I take it as almost axiomatic that the humility of scientists is such that as a class they will guarantee nothing. Human knowledge is vast but the unknown is infinite and the use of this slogan but reflects the layman's difficulty in comprehending the mental values of the scientist. The zenith of this argument, as an objection to fluoridation, was strangely enough reached by Dr Polya who submitted the following statement:—[1]

> 'We may grant that continued intake of fluoride in daily doses of 1-2 milligram has no major effect on the incidence of cancer and vascular diseases or on mortality statistics without accepting its safety in the absence of tests concerned with every possible organ and every possible disease: *a complete investigation of every individual is required rather than picked statistical summaries.*'.

The emphasis was that of the witness. He gave evidence on the same lines[2].

544. I say strangely because on cross-examination he admitted readily enough that he knew that to pose such a standard was to ask an impossibility but justified it by saying that this was his way of expressing his resistance to compulsion in the use of fluoridated water. I could have done with more chemistry and less casuistry.

545. Others were more reasonable. Professor Sir Arthur Amies[3] suggested that a 'reasonable medical certainty' was what was required, while Dr Sutton adopted the following quotation:—

> 'Professionals and specialists have the duty of insisting upon a scientific demonstration of a high probability that a proposed method will be useful and safe, before it is recommended for general adoption. The maintenance of this attitude is of paramount importance.'[4],

at which no one could cavil.

546. It is obvious that many regard this as a topic of importance. At the risk of prolonging a rather formal issue I would quote the remarks and conclusions of the South African Commission[5] with which I find myself in complete agreement:—

> '196. The argument has repeatedly been advanced that sufficient research has not yet been done to establish with *absolute* certainty that *no* single individual in the community will *ever* suffer harm as a result of fluoridation. In other words, opponents of fluoridation demand criteria for safety of the utmost stringency. Since such criteria would be impossible to fulfil, this argument turns upon a matter of principle rather than a matter of fact. In this matter of principle the attitude of orthodox medical science is quite clear. Where any therapeutic or preventive measure affecting the public health is contemplated, an estimate is made of the probability that it will do good and an estimate is made of the probability that it will do harm. The adoption or rejection of the measure then depends upon the balance of these probabilities. The absolute inevitability of benefit and the absolute impossibility of harm are, in the interests of public welfare, regarded as unreasonably stringent criteria . . .

'We have stated in Chapter 111 that we are entirely satisfied that the fluoridation of public water supplies will result in a significant diminution in the incidence of dental caries. We are equally satisfied that a satisfactory estimate of the likelihood of harm resulting from this procedure has been made and this indicates that it is so remote as to constitute no contra-indication to fluoridation. In conformity with the ethical and scientific principles that govern custody of the public health, we are agreed that attempts to satisfy demands for proof of absolute safety of the measure to every single member of the community would result in an unnecessary delay in the adoption of a beneficial procedure.'.

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CHAPTER 30. MORBIDITY AND MORTALITY COMPARISONS BETWEEN FLUORIDATED AND NON-FLUORIDATED AREAS

547. In the course of the fluoridation controversy it would be inevitable that close attention should be paid to studies in which morbidity and mortality rates for fluoridated and unfluoridated communities have been compared. Opponents of fluoridation have claimed that in some such studies significant differences are discernible between the two to the disadvantage of the fluoridated community. If they do not point inevitably to fluoride as the cause, nevertheless it is said, they constitute sufficient reason for delaying its introduction until further study may have either confirmed or dissipated their fears. These claims are the basis of an objection to fluoridation which it is necessary to consider.

548. Morbidity studies designed to determine the prevalence of specific diseases and to compare them between high-and low-fluoride areas are numerous. In relation to skeletal disorders many have already been discussed in Chapter 22 of this Report. It is not proposed to repeat that discussion. However, to the references there given I would add a reference to a report by Stevenson and Watson (1960)[1] which reviews 170,000 X-ray examinations of the spine and pelvis of patients (some at least of advanced age) from high fluoride areas. In twenty-three patients only, all of whom had lived their lives in areas with water fluoridated at levels from 4 ppm to 8 ppm, X-rays disclosed evidence of symptomless osteosclerotic changes. Other supporting studies mainly in relation to skeletal conditions but which also cover a wider spectrum of disease are collected in the Monograph 'Fluoride Drinking Waters[2, 3, 4, 5, 6, 7, 8]' and in the projected World Health Monograph 1967[9, 10].

549. An early and important study which has been the subject of controversy and which is really a morbidity study but which is often discussed in the context of mortality is what is known as the Bartlett-Cameron study[11, 12]. I do not intend to discuss it at length. It was perhaps the most thoroughly designed of all morbidity studies and was intended to compare disease rates between inhabitants of the two neighbouring towns, one with a water supply naturally fluoridated at 8 ppm and the other at 0.4 ppm. Unfortunately the samples were small, 116 subjects from Bartlett and 121 from Cameron and the age range was considerable, 15 to 68 years. A complete medical examination was made in 1943 and again of the same subjects in 1953. The authors went to some pains to trace those who had moved and died in the meantime.

550. From this study the authors concluded:

'The incidence of specific systemic abnormalities and the prevalence of abnormal laboratory findings showed no tendency toward higher or lower rates for either town, except that the number of cases of dental fluorosis was significantly higher in Bartlett than in Cameron and the difference in the rates for cardiovascular abnormalities reached a significant level in Cameron.'.

551. The criticism[13] of the study is mainly related to the smallness of the groups studied and as to whether differences noted in relation to specific disorders are statistically significant or not. Further, the age distribution of the groups was not identical (the Bartlett group being older than the Cameron) and this creates difficulties in interpreting the data in relation to ailments, the prevalence of which would be normally greater in the older age group.

552. I think it is right to acknowledge that despite the impressive thoroughness with which the study was planned, the groups studied, in the light of the spread of age and the range of disease are perhaps too small for positive conclusions. However, the trend is unmistakably in favour of the relative safety of fluoridation even at 8 ppm. In this context it may be recalled that Roholm[14] found that the morbidity of cryolite workers, measured by the number of sick days taken in the year, scarcely exceeded the average of industry as a whole and that 'There is nothing to show that working with cryolite reduces the period of life or that cryolite workers acquire macroscopically recognisable organic lesions apart from the bone changes'[15]. This is a significant comment because in the industry as he studied it he estimated that the daily rate of ingestion was of the order of 140 mg of cryolite equivalent to 70 mg of fluoride of which 15 mg would be absorbed[14]. This would exceed greatly normal ingestion rates in Bartlett.

553. If differences did exist in the prevalence of disease as between fluoridated and non-fluoridated communities, differences in mortality would only be found in respect of those diseases or morbid conditions which act to shorten life. However in small samples in particular crude statistics are of doubtful use. While statistical techniques may be relied upon to take care of chance variation; variations due to age, sex, race, occupation, geography and even such matters as the presence of specialised institutions caring for the aged or the chronicaly ill may mislead unless allowed for. This makes the interpretation as well as the planning of such studies, hazardous and, of course, inevitably provides grounds for controversy.

554. The Bartlett-Cameron study furnishes as good an example of this kind of controversy as any. The plan of the study called for an examination of the subject populations in 1943 and again in 1953. Within that period some in each group died. Table XXVII shows numbers examined and their grouping according to age and sex. It is clear that, assuming all other factors were equal, the age distribution within the sample populations of the two towns was unequal, Bartlett being predominantly older. The authors claimed that 'the difference between the age-corrected mortality rates is not significant by statistical analysis' i.e. over the whole subject population. Dr Exner, on the other hand, takes the sub-group 50-64 years of age and points to the fact that out of forty-five subjects there were five deaths during this ten-year period for Bartlett as against one out of fifty-five for Cameron, and refers to it as a six-fold difference in death rate[16]. The cause of the deaths is not stated. As Dr Exner says, 'maybe they were all run over by street cars'. Personally I am unable to ascribe any significance one way or the other to such a few instances in such a small sample.

555. The overall figures for deaths were re-examined by Committees of the House of Delegates of the American Medical Association in the course of a worthwhile report which repays study, and which report (1956) was subsequently adopted *in toto* by the House[17].

TABLE XXVII

DISTRIBUTION OF PARTICIPANTS BY LOCALITY, AGE, AND SEX, BASED ON 1953 AGE

Age	*Examined*, 1943			*Examined*, 1953			*Not*	*Deceased*		
Group	*Male*	*Female*	*Total*	*Male*	*Female*	*Total*	*examined*	*Male*	*Female*	*Total*
					BARTLETT					
25-29	5	12	17	5	9	14	3	....	....	....
30-34	....	4	4	....	2	2	3	....	....	....
35-39	....	....	....	....	....	....	....	....	....	....
40-44	2	3	5	2	3	5	....	....	....	....
45-49	4	11	15	4	10	14	....	....	....	....
50-54	2	6	8	2	6	8	....	....	....	....
55-59	6	8	14	3	8	11	....	3	....	3
60-64	10	13	23	8	13	21	....	1	1	2
65-69	2	11	13	1	9	10	....	1	2	3
70 +	8	9	17	6	5	11	....	3	3	6
TOTAL	39	77	116	31	65	96	6	8	6	14
					CAMERON					
25-29	11	6	17	9	6	15	2	....	....	....
30-34	....	1	1	....	1	1	....	....	....	....
35-39	....	5	5	....	4	4	1	....	....	....
40-44	1	4	5	1	4	5	....	....	....	....
45-49	6	13	19	6	13	19	....	....	....	....
50-54	6	11	17	5	11	16	....	....	....	....
55-59	11	13	24	11	12	23	1	....	....	....
60-64	4	10	14	4	10	14	....	....	....	....
65-69	4	10	14	2	9	11	....	2	....	2
70 +	3	2	5	2	3	5	....	1	....	1
TOTAL	46	75	121	40	73	113	4	4	....	4

Taken from Leone *et al.*[11].

Using another statistical method they 'calculated the probability that chance determined the difference between the age-adjusted deaths in the two sample populations. The result is in fact above the significant level (1 chance in 20) used by the authors for their criterion. These odds are approximately 1 in 21 that mere chance determined the difference (P—0.048)'. While twenty-one to one exceeds the level of significance (twenty to one) below which (e.g., nineteen to one) it might be said that the differences were not significant and could be ascribed to chance, after reviewing the matter in the light of other evidence, the Committees concluded and the House of Delegates adopted their conclusion that 'No evidence has been found since the 1951 statement by the Councils to prove that continuous ingestion of water containing the equivalent of approximately 1 ppm of fluorine for long periods by large segments of the population is harmful to the general health'.

556. Bartlett-Cameron was a study of morbidity, not mortality. Causes of death are not recorded in the study. It relates to a population drinking water fluoridated at 8 ppm. Incidental to its main purpose it indicates by one statistical method to the degree represented by the margin between 21 to 1 and 19 to 1, that a difference in overall mortality rates between the two towns was not due to chance. The opponents of fluoridation have to that extent impugned its value. If it stood alone, it may be thought to raise questions that should be answered. But, though great attention was paid to it, like so many of the epidemiological matters discussed in this inquiry, it does not stand alone and the wood is not demolished by the subtraction of one tree.

557. I refer briefly to the following:—

(*a*) The mortality statistics for one year were examined in respect of twenty-two Illinois cities, eighteen having no fluoridation and four with more than 0.7 ppm. City populations ranged from fifteen to forty thousand. Standardised mortality rates were extracted for deaths from—

(i) all causes;
(ii) heart disease;
(iii) cancer; and
(iv) nephritis.

The four fluoridated cities were randomly distributed throughout the array, i.e. no correlation was discernible related to fluoridation[18].

(*b*) A comparison of mortality in thirty-two pairs of cities in Illinois, each of one naturally fluoridated and one non-fluoridated city, and an analysis of mortality before and after the institution of a controlled water fluoridation programme did not reveal any relationship between mortality experience and the presence of fluorides in drinking water supplies[19].

(*c*) An examination of mortality experience in five large cities before and after fluoridation was made by comparing the 1940 and 1950 mortality experiences with that of their fluoridation year. As the cities had fluoridated in different years this had the effect of giving between them 1, 2, 3, 4, and 5 years' experience of fluoridation. The study showed no significant difference[6].

(*d*) In the United Kingdom an early study (1944) by Weaver[20] relating to Tynemouth (0.25 ppm F) and South Shields (1.4 ppm F) showed no difference in mortality figures for heart disease, malignant disease, cerebral haemorrhage and nephritis. Variable differences were found in relation to pneumonia, bronchitis and influenza. There was a big difference in tuberculosis rates in favour of the the non-fluoridated area which also had a fairly consistently better record for neo-natal deaths. The factor responsible for these differences is not identified but socio-economic factors are indicated which may have played a part.

(*e*) In Great Britain the Ministry of Health reported in 1956:

> 'As an additional check, comparisons have been made in this country of vital statistics in high- and low-fluoride areas and the following rates have been studied: neo-natal and infant mortality rates; stillbirth rates; death rate from all causes; death rates from kidney diseases (nephritis and nephrosis); cancer (all forms); ulcer of the stomach; cardio-vascular diseases; cancer of the stomach; and cancer of the breast. In none of these were there any differences between high- and low-fluoride areas which could be interpreted as indicating any harmful effect on health; nor even a slight pointer to the need for any further studies to demonstrate this.'[21].

(*f*) In 1957 Chalmers Clarke reported on a small village group in the county of Kesteven, a naturally fluoridated area in England. He said[22]:

> 'One small community had a public water supply naturally fluoridated at 2.5 ppm for twenty years. The latest mortality rate for that community was 10.5 per thousand of the population, compared with 11.7 for England and Wales and 11.7 for the county of Kesteven. There was nothing unusual about the certified causes of death. The ages at death however, showed a high expectation of life, for five of the 18 deaths occurred between the ages of 50-70 and 13 occurred between 70 and 90 years of age. There was, also, a considerable number of aged people alive in the area, eight being in the 80-90 year age-group and two aged 94 and 97, respectively. One man, aged 80, worked actively as a bricklayer. All had been consuming fluoridated water at two-and-a-half times the recommended concentration, without any evidence of ill effects'.

(*g*) In 1962 it was sought to duplicate the study mentioned as (*b*) above in the United Kingdom. Thirty-six areas were chosen consisting of eighteen with naturally occurring fluoridation (5.8 to 0.7 ppm) matched with eighteen non-fluoridated areas (less than 0.2). Difficulties were encountered in the matching of controls and are discussed. Nevertheless this is a wholly impressive study. The death rates adjusted for age and sex were used over a period of ten consecutive years. In the detail of the discussion and the care which the authors take in dissecting the raw data, there is ample justification for their summarised conclusion[23]:

> '1. An investigation has been carried out to see whether there is any relationship between naturally occurring fluoride in water supplies in England and Wales and mortality from all and from certain causes.
>
> '2. The difficulties of an investigation of this nature on British data are discussed.
>
> '3. The results indicated that the over-all mortality was the same in the fluoride and control areas.
>
> '4. With certain specific causes of death differences reaching the 5 per cent level of significance were found between high and low fluoride areas. These were conflicting and it was considered very unlikely that fluoride was the cause'.

Their conclusions were adopted by the Research Committee of the United Kingdom Ministry of Health in its 1962 Report[24].

(*h*) Comparative mortality rates in fluoridated and non-fluoridated areas were studied in Canada at the request of the Ontario Committee of Inquiry. Eighteen selected municipalities were used with water fluoridated at 1 ppm or greater and they were compared with nearby municipalities having a high fluoride concentration of 0.4 ppm or less. I quote the significant portion of the conclusion drawn[25]:

> 'Recent mortality data have been examined for eighteen pairs of "fluoride" and "non-fluoride" municipalities in Ontario and for the three cities, Stratford, Brantford, and Sarnia. Comparisons of mortality from all causes, of still birth rates and of infant mortality rates have been made. Mortality from cardiovascular diseases and cancer has been compared for those municipalities for which vital statistical tabulations are available in sufficient detail to permit the computation of age-adjusted, cause-specific rates. A consistent pattern emerges from these analyses. The variation in mortality is greater within the fluoride and non-fluoride groups of municipalities than between them. In some instances the fluoride municipality has the higher rate of mortality while in an almost equal number of instances the non-fluoride municipality has the higher rate. Furthermore differences, between pairs of municipalities bear no relation to the concentration of water-fluoride in the "fluoride" member of the pair.
>
> 'This pattern indicates that the mortality rates under consideration are not influenced by the fluoride concentration of the water supply.'

(*i*) Similar results are claimed for studies conducted in Russia[26] and Switzerland[27].

558. The studies referred to do not support any suggestion that fluoridation has or could have an adverse effect on morbidity or mortality. The great preponderance of the evidence is to the contrary.

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CHAPTER 31. OTHER DENTAL EFFECTS—PERIODONTAL DISEASE

559. The periodontal tissues are the alveolar bone of the jaws, the gums and the fibrous membrane which surrounds the root of the tooth and joins it to the alveolar bone. Inflammation and infection in these tissues—periodontal disease—is the other great cause of tooth mortality. In fact in the adult population more teeth are lost as a result of periodontitis than from any other cause, including caries. An American survey has estimated that in women over thirty-nine years

of age and in men over thirty-four years periodontal disease was the reason for between two or three times as many extractions as caries.

560. It may range from a mild gingivitis or inflammation of the gums, usually provoked by local irritants such as food debris and calculus or tartar to frank pyorrhoea in which there is an acute suppurative infection of the whole of the periodontal tissue. An association with increasing age is recognised but the condition is largely ascribed to neglect, in that maintenance of good oral hygiene is probably the most important measure for its avoidance[2].

561. In considering allegations of increased periodontal rates as due to fluoridation, one elementary but important fact must be kept in mind. Periodontal disease will not occur in edentulous persons who have lost their teeth. If therefore as a result of caries a person has lost his teeth by the age of twenty, his chances of subsequently suffering from periodontal disease are nil. On the other hand, the more teeth he has retained on entering adult life, the more he has at risk and the more significant could be his contribution as a statistic to the prevalence and incidence rates for this disease. That this may be so does not mean that fluoride is a direct cause of periodontal disease, whatever be its incidental effect in preserving teeth beyond the age at which they would normally be lost to caries only to fall victims to another disease which care and good oral hygiene may have prevented.

562. Some reliance[3] has been placed on a statement by Dillon (1953)[4] to the effect that in populations using a fluoride water containing ' a toxic part ' . . . ' periodontal disease will occur prematurely and a widespread loss of teeth at an early age will be the penalty for a percentage reduction in juvenile caries '.

563. There is really nothing to support this statement. I adopt the following comment that it:—

> ' . . . apparently rests upon a misinterpretation of the status of eight individuals examined in South Shields by Forrest, Parfitt, and Bransby, and an analytic procedure " which seems to be based on a misconception of the electro-chemical theory . . . Whatever " toxic part " may mean, the method of determining it is worthless " '[5].

and, after considering the comments of three expert witnesses[6, 7, 8] in whom I have considerable faith as to the competence of Dr Dillon in the field in which he writes, I am satisfied that the statement is not one of authority and need not be pursued further[9].

564. However, Sir Arthur Amies in his submission[10] made the statement that ' Some authorities have published observations that suggest strongly that the toxic effect of fluoride in water is associated with a higher incidence of periodontal disease ' and he cited two articles, one by C. C. Bass of (1957)[11] and the other by K. F. Box of (1953)[12]. Dr Sutton after referring to some early observations including one by Brown *et al.*[2], which I will mention later, referred to K. F. Box as well as to a statement by the late H. K. Box (father of K. F. Box) in the same year[13].

565. The latter statement (by H. K. Box) or the article[13] in which it appears has been the occasion of a criticism made by Dr Sutton[14] of the findings of the New Zealand Commission[15] and partial references to the same article are still being made in the current anti-fluoridation literature. Because of the habit, seemingly inveterate in this controversy, of encapsulating statements which some partisan (I am not referring to Dr Sutton but to others) thinks might be capable of some significance irrespective of withdrawals, retractions or even contradictions that may have later occurred, it is necessary to go over the ground again though it has been done many times before.

566. Professor H. K. Box, who is acknowledged to have been an authority on periodontal diseases, was reported to have said that ' where fluoridation is present naturally the prevalence of gum disease is very high '. In fact for reasons given at length by the New Zealand Commission[15] he never made any such statement. On the contrary in the article[13] already cited he said:—

> ' I have never made a survey of gingival and periodontal diseases in any area where the water was naturally fluoridated . . . and I have written or published nothing on this subject.'

567. This statement was quoted by the New Zealand Commission as a refutation of an alleged statement claiming detrimental effects as a fact. The Commission has been criticised by Dr Sutton[14] for not going on to add the following further statement, which, as a warning, by Dr Box appeared in the same article:—

'At the present time, the available findings on gingival and periodontal diseases, as revealed by survey, are totally inadequate. It is my considered opinion that the artificial fluoridation of water supplies, on a wholesale basis, should not be advocated or adopted until fully sufficient findings show that there are no harmful sequelae from a gingival or periodontal standpoint.'

and Dr Sutton has commented in his submission:— 'Little investigation has been carried out since that time'.

568. At the time that the statement was made (1955) there was at least one study in which some of the matters referred to had been considered. I have mentioned the 1954 study by Brown *et al.*[2]. Basically it was a study to evaluate the worth of an epidemiological index of gingival disease, but the authors in doing so observed the prevalence of gingivitis in groups of children from three Canadian cities. They said in their Report:—

'The findings of this study reveal the existence of an inverse correlation between the prevalence of gingivitis and oral-hygiene status. The results of the chi square test show this relationship to be significant in all three population groups under study. The variation in the proportions of children affected in Brantford, Sarnia, and Stratford is a reflection of oral-hygiene status in the three cities.

'It is worth noting that these three cities furnish the study samples in a study of water fluoridation. Stratford water contains fluorides naturally at a level of 1.3 parts per million. Fluoride has been added to the Brantford water for a period of approximately eight years to obtain a concentration of between 1.0 and 1.2 parts per million. Sarnia's water supply is fluoride-free. There appears not to be a significant relationship between the presence of fluoride in the water supply and the occurrence of gingivitis. Gingivitis prevalence and severity were found to be equivalent in Brantford, where the water is fluoridated, and in Sarnia, where the water is fluoride-free. One might speculate that the high incidence of gingivitis in Stratford children reflects a public carelessness in respect to oral hygiene, attributable to low caries rates of long standing.'

569. In the same year Zimmerman *et al.*[16] published a study in which they reported the results of an examination of adult residents of Bartlett (8 ppm) and Cameron (0.4 ppm) repeated at an interval of ten years. A wide range of dental abnormalities were surveyed and both clinical and roentographic examinations indicated that there were no significant differences in the periodontal health of the inhabitants of the two towns. Since that date there have been three major studies[17, 18, 19] conducted by Englander and his associates comparing the periodontal health of the inhabitants of Aurora (naturally fluoridated at 1.3 ppm) and Rockford (0.1 ppm Fluoride). They covered three age groups, teenagers (aged 13 to 17), young adults (aged 18 and 19 years) and adults (18 to 70). Differences between the two cities were slight but such as did exist were, generally speaking, in favour of the fluoridated city. I quote the findings as to the adult group[18] which may be taken as typical:—

'1. Although the prevalence and severity of periodontal disease was high in both cities, lower periodontal indexes (PI), indicating somewhat less severity of disease, were found at each age level in continuous residents of the naturally fluoridated city. For all ages combined, the difference between periodontal indexes for the cities was statistically significant ($P.<0.01$).

'2. The percentage of persons with one or more frank periodontal pockets was significantly higher in Rockford.

'3. There was no difference in the proportion of persons free from overt periodontal involvement between the cities.

'4. Oral hygiene scores were about the same in Aurorans and natives of Rockford. Calculus was equally present, indicating that the continuous consumption of optimally fluoridated water neither promotes nor inhibits calculus deposition.

'5. Differences in severity of periodontal disease may be due indirectly to disparity in caries rates between the two cities. Fluoridated water may also have a deterrent influence on the progress of destructive disease that has become established.'

570. These are substantial and not slight studies. There has also been a 1957 study by Russell[20] in which the periodontal status of the residents (children and adults) of three fluoridated communities was compared with that of the residents of three comparable low or non-fluoride communities. Ages of the persons studied, who numbered nearly 20,000 ranged from seven to seventy-eight years. What differences were found were in favour of the fluoridated populations

but they were not regarded as significant. Again I would think that the conclusion may be left to speak for itself:—

> 'Most of the differences were slight. They are not adequate to support a hypothesis that use of a fluoride water results in improved health of periodontal tissues. The findings are, however, wholly incompatible with any hypothesis that the periodontal tissues of children or of adults are harmed by use of a fluoride-bearing domestic water.'

571. Russell and White also reviewed the whole matter in a separate chapter of a monograph which appeared in 1959[21].

> 'The only conclusion consistent with the data is that use of a fluoride domestic water has no effect, for better or for worse, upon the health of gingival and periodontal tissues.'

572. In Holland Houwink and Backer Dirks[22] in connection with the Tiel-Culemborg trial made a comparative investigation of dental calculus formation between children of the two cities after eight years of fluoridation in Tiel. A slight but not a significant difference was found, the higher degree being in the fluoridated city. Their conclusion was:—

> 'Owing to the fact that the calculus distribution is very skew and the standard deviation extremely large, it is difficult to establish whether the two cities differ significantly in respect of the calculus frequency of the children. Even though a large number of children was examined in each city doubt remains as to whether there was a real difference between them. A higher calculus score might have been due to the presence of a greater number of abutting teeth. Thus, fluoridation of the piped water supply may indirectly—through reducing the number of extractions—increase the calculus scores by a small extent. If there is any direct effect, it is small.'

Adler refers to studies in other parts of Europe. He says they 'justify the conclusion *that the possibility of increased fluoride consumption having an adverse effect on the periodontium can be definitely excluded*'[23] (emphasis is that of the writers of the article cited).

573. In the light of these extensive studies and reviews I find it difficult to understand what weight should now be attributed to this much quoted warning by H. K. Box in 1955 bearing in mind that despite his eminence in the general field of periodontal disease he himself had never made a survey of the subject of his specialty in a fluoridated area and had written or published nothing on the subject. I can only report that the effect of the evidence to which I have referred, supplemented by the oral evidence that I have taken, is overwhelmingly to support the contention of the fluoridationists that periodontal disease is not influenced one way or the other by fluoridation, except possibly indirectly by lessening extractions and thus providing more teeth to attack, more contiguous surfaces in which to entrap detritus and possibly, though this is disputed by some, by reducing standards of oral hygiene as a result of the over confidence engendered by a caries free mouth.

574. The paper by Bass (1957)[11] to which Sir Arthur Amies referred I found somewhat extraordinary. The theory it propounds is that—

(*a*) Use of a domestic water with as little as 1 ppm of fluoride increased gingivitis activity;

(*b*) The resulting inflammatory exudate (pus?) inhibits dental caries particularly in the interproximal spaces; and

(*c*) The observed reduction in dental caries prevalence in populations using a fluoridated domestic water 'results entirely from the contemporary increase in periodontoclasia (gingivitis) activity.'

It would perhaps be sufficient to dismiss it by referring to the very faint support it received at his hands as evidenced by the following passage from the Transcript[24] in which I put some questions to Sir Arthur as to what weight he ascribed to it:—

> '4. Mr Commissioner . . . This reference that you have to BASS, I have read this paper. It seems to be rather a curious paper. I don't know what authority or weight you attribute to this particular theory. Do you agree with his suggestion? Let me refresh your memory—first of all it is a very personal paper; he had some theory about brushing teeth which he refers to as almost a personal invention, and he develops this at great length, and then he goes on to say that the decrease in caries in a fluoridated area is not however due to the action of fluorine but it is due to the increase in periodontal disease in the same period, which by reason of the toxic or septic exudate of the periodontal disease, inhibits the surface caries. Do you accept that theory? . . . As a theory, yes.

'5. I am not quite clear what you mean by that. Now, what do you mean? Do you mean that your clinical experience makes you feel there is any probability in that theory? . . . To this extent, that it is well known that, while they are not actually exclusive dental caries and pyorrhoea generally are found more marked—more caries and less pyorrhoea; more pyorrhoea and less caries.

'6. We discussed this a minute ago, and we agreed there was an age difference. Do you say it is probable that caries deficiency in children under the age of twenty, found in a fluoridated area, is due to the increase in some children of periodontal disease? . . . No necessarily.

'7. That means you don't accept his hypothesis? . . . I accept his hypothesis as an hypothesis, but I would not strongly substantiate it.

'8. I still find some ambiguity in that? . . . I will try and answer it directly.

'9. I am still not getting exactly what you think of that, not just as an hypothesis but as to its probability; as to its acceptability to you as a practising dentist? . . . Well I would say—trying to be perfectly frank—I cannot disprove it, but I would want a lot of proof for it. Does that answer your question?

'10. Well, by reading between the lines to some extent it does? . . . I am trying to be truthful.

'11. I wondered why you mentioned this particular article? . . . Because he is one who has written in this way.

'12. That is my point over and over again. Does it help me merely to throw these at me unless you are prepared to say, "I approve of this; I agree with it; it expresses my own opinions;" but this one doesn't? . . . Not strongly, no.

'13. I will leave it at that.'.

575. But the theory has been examined in detail point by point by Russell and White[21]. Their detailed refutation by reference to precise epidemiological data is entirely convincing and I accept it . I refer also to the discussion of this matter by the New Zealand Commission and to the evidence which they detail[15].

576. The last paper to which I must refer in this connection is that of K. F. Box[12] which is a series of editorial speculations and theoretical inferences related to no specific experimental or epidemiological data. From one aspect it seems to propound a view of the fluoride content of dental plaque which is inconsistent with modern work already referred to, from others it is simply not confirmed by the specific studies I have mentioned earlier. With great respect I would not at this time of day think that it should be considered as in any measure authoritative.

577. There is no evidence of any direct causal relation between fluoridation at 1 ppm and periodontitis. All the evidence points to increasing age and faulty oral hygiene as the significant factors in its prevalence and the papers and observations cited do not furnish any argument of substance against fluoridation at optimum levels. Indeed, such indications of differential effect as exist tend to favour fluoridation rather than otherwise[23].

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CHAPTER 32. FLUORIDE AND CELL METABOLISM

Section (a)—Cancer: Dr Alfred Taylor

578. In this chapter I propose to consider briefly a skein of evidence and articles which are concerned with *in vitro* and other experiments designed to test the effect of fluoride at varying concentrations on mammalian cells both normal and neoplastic, and the inferences that are sought to be drawn from them.

579. The general subject of *in vitro* experiments is referred to elsewhere (Ch. 36, *post*). The danger of directly extrapolating results so obtained to the living human body must always be borne in mind. One aspect of this danger which has a special significance in relation to the matters to be discussed in this section is concerned with the ability of the living body to maintain the fluoride concentration of the human plasma at a much lower level than that which may be present in food or water consumed[1, 2]. (See Chapter 9 (b), *supra*).

580. The suggestion that fluoride can be a cause of cancer and be detrimental to the normal growth and reproductive processes of mammalian cells is based on two series of experiments, one conducted by Dr Alfred Taylor of the U.S.A. and the other by Drs Berry and Trillwood of England, coupled with a remarkable document which I will call for easy reference 'The Time Lapse Film'. It was produced to the Commission by Dr Hale of Hobart but originates in America.

581. I do not intend to describe Dr Taylor's experiments[3, 4] in detail. He claimed that water fluoridated at low concentrations accelerated the progress of cancer in a strain of cancer susceptible mice (96% to 100% mortality[5]). Unfortunately his initial experiments overlooked the content of the solid food (42 ppmF^-) fed to both test and control animals. In later experiments he sought to rehabilitate and enlarge his observations, until he arrived at the conclusion that drinking water fluoridated at 1 ppm shortened the life span of cancer-disposed mice by 9% whether they died of cancer or not[6, 9]. The criticism they have attracted[7, 8] has been so complete that there is no point in dwelling on them.

582. They have been completely controverted on all major points by other experiments using better techniques[7]. Not the least of the many points of criticism is the fact that the amount of fluoride claimed by Dr Taylor to produce maximum tumour growth (0.000,005 mg) when injected, is so small in relation to the normal fluoride content of mouse plasma (0.1 ppm) that it would be quite incapable of producing any fluctuation in plasma concentration beyond normal variation[7]. In such circumstances it is difficult to see what foundation there could be for the effect claimed. In relation to longevity other researchers found no significant difference between mice drinking unfluoridated water and fluoridated water although in their case the mean age at sacrifice (on developing cancer) was in both cases longer than the whole life span found by Dr Taylor for either of his groups[7].

583. But in fact it would not be right to ascribe to Dr Taylor the claim that fluoride causes cancer. He himself has disowned such an assertion[9] limiting himself to the statement that it accelerated its development[6, 12]. The claim is constantly made however in anti-fluoridation litera-

ture and his work is cited. Support for the same assertion is to be found in the work of Professor Steyn (1964)[10]. Professor Steyn's theory was examined by the South African Commission, which discusses the matter technically and at length. I quote one paragraph[11]:

> 'Steyn quoting Holman and apparently agreeing with him, suggests that fluoride may inhibit catalase and that this may in some way cause cancer. There is no substance in this belief. Tumours have not been found to differ qualitatively from normal tissues in their enzyme content. There is no "cancer enzyme" which appears only in neoplastic cells, nor is there a systematic deletion of any enzyme or group of enzymes which distinguishes between tumour and normal tissue.
>
> 'Catalase is widely distributed in living cells and while its activity is often decreased in cancer cells, this is the effect of neoplastic transformation and not the cause. No one has ever produced a cancer by inhibiting catalase only.'.

584. The prevalence of cancers of various kinds has been made the subject of specific study in relation to mortality rates in the U.S.A., Great Britain and Russia. No correlation with fluoride has been found[13, 14, 15, 16, 17, 18, 19, 20].

585. As Dr Taylor's work related to mammary cancer in mice, of particular interest is the observation by Hodge and Smith[5] that in New England, where water supplies contain little or no fluoride, the prevalence of cancer of the breast was 17 per 100,000, whereas in Texas, a fluoride-rich State where water contains up to 5 ppm, the average rate was only 7 per 100,000. I class this as an observation only, not of course as a proof of any cancer inhibiting quality, but it may I think justly be set alongside any fears sought to be engendered by reference to Dr Taylor's experiments on mice 96 to 100% of which die of this form of cancer anyway. I note too that before the Irish High Court in the case of *Ryan* v. *Attorney-General*[21], which ranks as a *cause célèbre* in the fluoride controversy, the suggestion that fluoride caused cancer was expressly disclaimed by counsel who appeared for the objectors, presumably because those who instructed him, and they included some whose names still figure prominently in this controversy, were unwilling to assert it as a fact. It could be for this reason that the subject is ignored in the 1967 monograph being prepared under the editorship of Professor Ericsson for the World Health Organisation. This proves nothing of course, but it might have indicated a need for caution on the part of those who sought to advance the matter in these proceedings.

586. Of interest in this connection is the fact that in humans large doses of fluoride have been used therapeutically in an effort to arrest cancerous growths. Black *et al.*[22] administered up to 320 mg a day of sodium fluoride to a group of seventy patients for periods of five to six months without any evidence of acute or chronic toxicity. Doses of up to 600 mg given intravenously to patients with leukaemia were tolerated without deleterious effects. He concluded however that malignant tissue might show a greater sensitivity than normal tissue to such dosages.

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Section (b)—Fluoride as a Protoplasmic Poison: Drs Berry and Trillwood

587. The sensitivity of malignant tissue to large doses of sodium fluoride noted by Black *et al.*[1] is perhaps more in accordance with the result of the second study which has been referred to in this context of cancer and cell metabolism. In 1963 Berry and Trillwood in a letter in the British Medical Journal[2] reported some *in vitro* observations on the effect of solutions of sodium fluoride on two tissue cultures, one a culture of human cells from a cancer of the cervix, and the other a special strain of connective tissue mouse cells. Both cell lines had been maintained in culture outside a living host for many years. It was claimed that a concentration as low as 0.045 ppm fluoride (or 0.1 ppm NaF) depressed the growth rates of these cell cultures, though no significant reduction of cell reproductive capacity was detected up to 4.5 ppm (10 ppm NaF); however at 45 ppm fluoride (100 ppm of NaF) a lethal effect on the culture was apparent.

588. The authors were cautious in the conclusion to be drawn. They said[3]—

> 'These studies were performed in tissue culture under conditions where a known amount of sodium fluoride was present in the medium continuously throughout the experiment; any direct analogy to the situation in the human being where various amounts of fluoride would be ingested at irregular intervals would be precarious in the extreme. It is our feeling that while these studies show effects upon two types of mammalian cells in tissue culture at high dilutions of sodium fluoride, they do not indicate any known deleterious effect of sodium fluoride ingested from drinking water fluoridated at the recommended level of one part per million.'.

Later the senior author said in an advice[4] to the late Mr Geo. Gray, M.H.R., who produced it to the Commission:

> 'I simply do not know whether these results can be extrapolated in any meaningful way for the assessment of a potential hazard to a human population from the chronic ingestion of fluoridated drinking water.'.

The same caution and understanding of the limited application of such *in vitro* experiments has not been shown by those who have cited the results of these experiments as proof that water fluoridated at 1 ppm is a 'protoplasmic poison' and can kill or damage human cells[5].

589. *Prima facie* there is an antipathy between the results claimed by Taylor and those indicated by Berry and Trillwood and this has been commented on[6]. Any purported analogy between the metabolic processes of cancer cells and normal human cells would ordinarily be suspect particularly in relation to their capacity for growth[7]. However other investigators who sought to duplicate the experiments have refuted the observations of Berry and Trillwood and their techniques have been authoritatively discounted[8, 9].

590. Armstrong *et al.* (1965)[10] duplicated their experiments and extended them to other tissue cells as well. They also sought a more thorough testing by seeking determinations in relation to additional parameters indicative of cell multiplication and vitality. At the concentrations used by Berry and Trillwood they found no relation between the amount of fluoride in the culture media and the rate of cell multiplication. Extending the experiments they found no significant differences between fluoride enriched solution and saline controls up to 15 ppm of fluoride (this is at least 75 times greater than normal plasma levels) where a doubtful but possible difference was noted in the protein content of the cells studied. They concluded from other experiments that Berry and Trillwood's results may have been due to faulty techniques in relation to the glass surfaces on which the cells were cultured. Their conclusion was—'It seems certain that the fluid environment of cells (i.e., in a human host) cannot reach the fluoride concentrations which are required to inhibit cell growth of those cells which have been studied under *in vitro* conditions.'.

591. Other investigators have studied other cell types and support these conclusions[11, 12, 13]. It is interesting to compare these *in vitro* experiments with other experiments on animals in which intravenous injections of up to 34 mg of sodium fluoride per kg of body weight (equivalent to approximately 1 gm in the human) have been given to rabbits daily for ten days without apparent systemic or toxic effects[1].

592. These matters were discussed with Professor Thonard in particular and I accept his conclusion that the papers lastly referred to are acceptable scientific evidence to show that in order to inhibit mammalian cells in tissue culture it would appear necessary to reach a concentration of at least 10 ppm of fluoride. This in relation to values capable of being found in human plasma is so excessive and so far exceeds experience as to be incompatible with life[14].

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Section (c)—Time Lapse Film

593. The third head of evidence offered in this regard was what I have called the 'Time Lapse Film'. This was a film of a mouse cell culture taken by a technique in which single frames of a continuous cinematographic film are exposed at intervals over a long period of time (in this case half-minute intervals for two weeks). The completed film when run off at normal speeds gives a graphic representation, magnified in time, of changes which would otherwise be imperceptible to the observer.

594. The film, prepared and published by the 'Greater New York Committee Opposed to Fluoridation Inc.', purports to show 'the effect on living cells of fluoride in concentrations far lower than in fluoridated drinking water—only thirty-eight thousands of one part per million to be exact'[1], i.e., .038 ppm fluoride. It is preceded by an elaborate introductory address[2], then there is another shorter introduction and a running commentary[3], purporting to explain for the layman aspects of cell metabolism and constitution relevant to the matter under discussion. Specifically it explains that mouse cells have been employed because they are mammals and permit analogies to be drawn in relation to humans. It purports to demonstrate the lethal effect, after an initial period of stimulation, of fluoride at the concentration mentioned on the living culture.

595. What the introductory address, the introductions, the commentary and the film itself do not disclose or even hint at is that at the same time as this film was made a similar film and study was made of two human cell cultures (tooth pulp and heart tissue) which showed no deleterious effects at all. This is all the more surprising in that the introductory address states a need for research into the action of fluoride on the metabolism of heart cells. The suppression of this important aspect of the so-called experiment was protested by the Director of the Laboratories in which the film was prepared[4, 5, 6, 7] in the following terms[6]—

> 'In my personal opinion there appears to be a very significant difference in the results shown in the pictures between the effect of the sodium fluoride on mouse and human cells and that the showing of the mouse cell pictures only is omitting vitally important information and grossly misleading.

'In view of these premature and unauthorised showings of only a part of the results of the mouse cell pictures and intentionally withholding the most important pictures of the effects of the fluoride on human cells, it has become necessary for the Time-Lapse Research Foundation as well as myself as Director, to withdraw from any further participation in this project.'.

596. Two documents alleged to be 'postscripts'[8, 9] to the film, supplied to the Commission, one by Dr Hale and one by Mr Kausman, purport to excuse the omission of the human cell experiments, but for different reasons. A suggestion in the second 'postscript' that the work was not acceptable because of 'lack of proper specific laboratory controls' is refuted by the Director of the Laboratory, nor is it explained how in any way they differed from those applicable to the mouse culture. Both 'postscripts' are undated but it is obvious they are not documents contemporaneous with the film, they are not part of the film track and have obviously been prepared in answer to criticism which could only have been made after the film was released.

597. In the absence of information as to the simultaneous experiments with human cells, (of which I was not aware when I first saw the film), as the Director of the Laboratory implies in his protests, the film would be a *suppressio veri* and a fraud on the public. The attempted suppression of these material facts robs the film of any character as an objective scientific experiment and I think it unnecessary to discuss its dubious merits any further, except to remark that even if the mouse culture be regarded as an experiment in its own right, reasons exist as detailed in the evidence of Professor Thonard[10, 11] for rejecting its purported conclusion.

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CHAPTER 33. THE KIDNEYS

598. The role of the kidney in concentrating fluoride preliminary to excretion has already been discussed. Retention and storage does not occur as concentration is followed by urinary clearance[1]. In fact it would seem that both in health and disease the kidney is remarkably tolerant to concentrations of fluoride far in excess of those relevant to fluoridation at 1 ppm. It is suggested that this may be due to the fact that its excretory mechanism would seem to depend mainly on glomerular filtration and little on tubular reabsorption enabling it to excrete fluoride at least 100 times more effectively than chloride[2, 3, 4].

599. There is no evidence that fluoridation at 1 ppm will have any deleterious results on the human kidney. The only study[5] (on rats) that may be thought to have indicated otherwise has been so discounted that it can be disregarded[6, 7, 8, 9, 10, 11]. From a large body of animal data it has been calculated that the minimal fluoride concentration in the drinking water to cause kidney injury on repeated administration is 100 ppm[12] but even larger doses for limited periods have been tolerated without ill effects (*ibid*).

600. I refer again to the discussion of this organ both in health and disease under Part 'H' dealing with the 'Abnormal Individual' (Ch. 25, Section (b), *supra*). I am satisfied that no useful purpose can be served by taking the matter any further.

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CHAPTER 34. THE THYROID GLAND

601. An objection that frequently recurs, particularly in the earlier literature, is that fluoride is an iodide antagonist and as a result has goitrogenic properties. No witness appeared personally to support this suggestion by direct evidence and it was refuted by the only technically competent witness who referred to the matter. Others however relied on the literature and as Tasmania has a history of endemic goitre I think it is proper briefly to indicate my reasons for dismissing it as an objection.

602. The literature is extensive and technical. Much of it is in foreign languages and the general subject of the thyroid gland, whether normal or pathological, does not seem to be one which admits of dogmatism. However it seems to be clear that in relation to this gland and its function there is no proof anywhere of detrimental effects to human beings caused by drinking fluoridated water. There is a great deal of epidemiological evidence the other way to which a brief reference will be made presently. But opponents of fluoridation argue that there are sufficient uncertainties in the present state of our knowledge as to the interrelationship of ingested fluoride, iodide, the secretions of the thyroid gland and other hormones to raise a presumption or at least a suspicion of possible harm.

603. There seem to be two general premises underlying the assertion. The first is that fluorine, like iodine, belongs to the elemental group of halogens. The thyroid gland is known selectively to accumulate iodide which it uses in the synthesis of its essential hormone thyroxine; there is said to be some evidence that to a lesser extent it may take up bromide and chloride (fellow halogen derivatives), hence the suggestion follows (and I quote from one of the most authoritative papers[1] in which the matter is advanced):

> 'It is a reasonable assumption that fluorine will behave fundamentally in the same way.'

604. The second premise derives from the fact that for a period of at least thirteen years, from approximately 1930 to 1943, fluoride or organic compounds of the element were used therapeutically to combat hyperthyroidism with in some cases at least beneficial results. In fact, Roholm[2] records that the treatment had been instituted as long ago as 1881. Hence the suggestion that there is an antagonism between thyroxine and fluoride[1] or between fluoride and iodide[1] without which the thyroxine molecule is physiologically inert. These two matters in combination lead to the further suggestion that in the normal subject fluoride as an iodide antagonist may lead to a hormonal deficiency and ultimately to goitre.

605. The argument thus summarised finds its main support in the writings of Gordonoff and Minder[1] and Steyn[3]. The latter claims that he was prompted to inquire as to a possible relationship between fluoride ingestion and goitre by the large number of cases of thyroid enlargement that he saw in the North West Cape Province of South Africa in 1936 including the Kenhardt Reserve. He claimed that in the school children in Kenhardt (water supply 3.2 ppm F) there was 'a fair percentage of simple goitre with the higher incidence in girls above the age of ten years'. The matter of 'Kenhardt Disease' has already been referred to (Ch. 22, Sec. (e), *supra*). It is difficult to accept this observation as meaningful in the light of subsequent detailed investigations of the Kenhardt children by Jackson[4] and by the two special investigators appointed by the South African Commission[5] who found exactly the reverse, viz. not a single case of thyroid enlargement in any of the 300 children examined.

606. As to the first suggestion that the thyroid gland may take up the halogen fluoride in substitution for iodide and use it in the synthesis of hormone (or as it is sometimes put that it is 'Halide-blind') modern studies have failed to show any correlation between the fluoride content of the gland and the level of fluoride intake either in normal subjects[6] or in those with hyperactive glands[7, 8], or in tests made with the aid of radioactive fluorine in both humans and animals[9, 10, 8, 11]. In the latter studies referred to no concentration of F^{18} in excess of that of the blood was found in the gland, though measured as concentrations (percentages of the F^{18} dose per gram of gland tissue) the values found exceeded those found for many other organs and tissues of the body except bone or the kidney[12]. The conclusion drawn is that the gland does not store fluoride in the sense of concentrating it as it does iodide, but reflects in its concentration the level of the blood and in its content the vascularity of the gland. This would seem to be confirmed by studies conducted in the 1960's[13] which suggest that not only is the gland not 'halide-blind' but that physio-chemical considerations involved in ionic size, valency and shape may prevent the fluoride ion from entering the iodide transport system.

607. Endeavours to ascertain by analysis the fresh tissue content of the gland have failed to show fluoride levels which do other than reflect the transitory levels of whole blood fluoride. They are reviewed at length by Hodge and Smith in 'Fluorine Chemistry'[12] and their conclusion seems unimpeachable:

> 'To summarise, the thyroid contains normally only trace quantities of fluoride when fluoride is ingested. The thyroid fluoride levels follow closely the blood concentrations; fluoride is not stored in the thyroid.'.

608. Higher values (from 10 to 30 ppm) have been obtained in analyses of the dried gland[14]. The maximum figure obtained by Call *et al.*[6] in a subject with a high fluoride exposure was 23.5 ppm dry weight as against the mean for the group to which the subject belonged of 5.2 ppm with a minimum of 0.3 ppm. These are values for fat-free tissues dried and ashed before analysis and cannot be applied directly to fresh tissue concentrations without allowance for fluid loss. If values are extrapolated roughly[34] they would seem to indicate a level higher than normal blood concentrations but it has been suggested that such isolated instances may be associated with centres of calcification in the gland[15, 16, 13]. Fluoride so deposited is of course pharmacologically inert and no significance has been attributed to such a finding[6]. It is to be remembered that as against this maximum value of 23.5 ppm, in the dried fat free gland, the normal thyroid contains some 4,000 ppm of iodide.

609. The South African Commission dubbed the whole argument a 'speculative assertion' and dismissed it summarily. For good measure it added in relation to the second limb of the argument:

> 'Moreover, the substitution of fluorine for iodine in the thyroxine molecule is not known to occur and is virtually impossible on theoretical grounds.'[17].

This is supported elsewhere[18, 13].

610. Nor do modern studies confirm the suggestion of earlier investigators (on which generally speaking Gordonoff and Minder rely) that an increased fluoride intake may diminish either iodide storage in the gland or the iodide level in the plasma, at least in relation to any concentrations relevant to fluoridation proposals[19, 20].

611. Paradoxically, however, there is evidence that in hyperthyroidism or a state of pathological activity of the gland, prolonged administration of large doses of fluoride has been known to exercise a beneficial effect. This was the rationale of its therapeutic administration for a number of years but the therapeutic action was apparently uncertain, weak and transitory[20] It has since been abandoned in favour of more reliable agents. I have found no explanation for

this apparent paradoxical effect in that fluoride may sometimes exhibit a specific action (in this case a beneficial one) in pathological states while being inactive in normal persons but apparently analogies are not unknown[20]. It does not follow however that in the normal subject deleterious results will follow as a result of the ingestion of fluoride even at raised levels and they have not been demonstrated (*ibid.*).

612. Gordonoff[1] makes the assertion that the frequency of goitres where fluorosis is endemic has struck many observers. He refers to three in particular. The first was Steyn[3] to whom I have already referred. The second was D. C. Wilson (1941)[21]. This was an early observation reporting a high degree of dental fluorosis in the goitrous area of the Punjab and a similar parallelism in an English county but goitrogenic factors were not investigated and the iodine intake was not studied. A subsequent study[22] in which the same author participated and which in Great Britain made a country-wide survey of thyroid enlargement failed to demonstrate any connection between goitre and fluoride concentrations in the water. Another reference was to an Italian paper which I have not seen (though I note that the Research Committee of the British Ministry of Health refers to opposing Italian observations[23]) but there are so many reliable observations (references are collected in the projected World Health Monograph[20] and in 'Fluorine Chemistry'[15]) to the contrary, some of which Gordonoff himself refers to, that I have not thought it necessary to pursue the matter.

613. Singh[24, 25] found no evidence of thyroid deficiency in his studies of chronic fluorosis in the Punjab nor did Siddiqui (1960)[26] nor did Roholm[27] (apart from one doubtful case in over sixty subjects examined) in his cryolite workers. Nor were they found, though looked for, in the Fort William Investigation[28]. In Switzerland a country with previously a high endemic goitre rate the matter has attracted much attention. Clinical examinations of children receiving sodium fluoride tablets for two years failed to reveal any thyroid changes and in a naturally fluoridated area (1.4 ppm fluoride) goitre has been effectively controlled by iodised salt notwithstanding the fluoride in the water[18]. In one experiment referred to by Professor Holdsworth patients with normal thyroid function were given from 3 to 5 mg of fluoride daily for up to fourteen months with no significant results[29]. In the morbidity and mortality studies referred to in Chapter 30 (*supra*) no correlation between thyroid deficiencies and fluoride intake was detected. A great number (in excess of fifty) of other references are collected by Smith and Hodge in 'Fluorine and Dental Health'[15] at p. 23 but it is unnecessary to repeat them. The observations of Steyn and Wilson are discussed in the New Zealand Report[30]. I entirely agree with the conclusion that they fail to demonstrate any causative connection between fluoride intake and goitre.

614. Gordonoff[1] refers to some early animal experiments in which pathological changes in the gland have been described following administration (generally by injection) of large (sometimes lethal) doses of fluoride compounds. References are not given but the experiments may I think be identified in Roholm[31]. They have no significance to fluoridation at 1 ppm. Reviewing a great deal of experimental data Hodge and Smith[32] conclude that the level of dietary fluoride which over a prolonged period can produce either microscopically detectable changes in the gland or measurable changes in function is 50 ppm. This opinion was adopted by the Expert Committee of the World Health Authority in its 1958 report[18]. Roholm himself experimented with rats and other animals and found no changes though other symptoms of toxicity were present nor did he in the two post mortems he conducted on humans with skeletal fluorosis nor have any such ever been described in other human post mortem studies from high fluoride areas.

615. A recent (1964) study by Leone *et al.*[33] should do much to allay anxiety on this score. Two groups were taken, one on 106 persons who used water naturally fluoridated at 3.48 ppm and another (control group) using water essentially fluoride free. All had been residents of their respective communities for more than ten years. Clinical examinations supplemented by determinations of protein bound iodide as a measure of thyroid function failed to demonstrate any significant difference between the two groups.

616. On the evidence taken and on the literature to which I have been referred or to which I have myself referred in an endeavour to elucidate the technicalities in which this subject abounds I can only report that I can find no reason for suspecting fluoridation at 1 ppm will affect the size or function of the thyroid gland.

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CHAPTER 35. THE CARDIO-VASCULAR SYSTEM

617. I heard no evidence specifically directed to the action of fluoride on the heart and the cardiovascular system generally. Steyn[1] refers to some observations of Takamori in Japan of a high rate of heart disorder in adults and children in a village where the fluoride content of the water ranged from 0.6 to 4.0 ppm. These observations were critically examined by the South African Commission and dismissed[2]. Among other comments they noted that the changes in the electrocardiogram which were reported and relied upon ' are not generally regarded as abnormal '.

618. A submission to the Commission by Dr Lilliendahl[3] an American opponent of fluoridation referred to some *in vitro* experiments on embryonic heart muscle with some other Japanese observations alleging myocardial damage to experimental animals fed large quantities of fluoride. In view of all the observations to the contrary from Roholm onwards and in view of the toxic quantities of fluoride employed, they seemed to me, as they have to other investigators[4, 5], to be irrelevant. I refer again to the mortality studies notice in Chapter 30 (*supra*) which clearly fail to indicate any correlation between fluoridation at 1 ppm and diseases of the cardiovascular system. The matter of metastatic calcification which might have been included under this head has been dealt with elsewhere (Ch. 39 and 43 (c) *post*).

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CHAPTER 36. ENZYMES

619. Enzymes are substances formed within the body which have a specific action in promoting chemical changes. The number so far identified in the human body runs into thousands, and they are an essential part of many vital processes.

620. A constant claim in the fluoridation controversey is that fluoride is an enzyme poison. The claim is based on *in vitro* experiments which demonstrate enzymatic reactions being inhibited by low concentrations of fluoride[1].

621. Apart from such laboratory experiments no clinical evidence is available to support the conclusion drawn by those who refer to them, i.e., that they constitute a valid analogy on which to found predictions for similar results in the human body. In other words, no such dire consequences are known or have been suspected in individuals drinking water fluoridated at 1 ppm or even higher. On the contrary, in those investigations in which serum enzymes have been tested following fluoride therapy at high dosage levels, no significant differences have been noted[2, 3].

622. Experiments *in vitro* have as analogies an intrinsic fault in that the conditions prevailing in a test tube will be qualitatively different from those that prevail in the living intact body. The homeostatic and biochemical mechanisms that within the body operate to govern and modify enzymatic reactions are not capable of accurate reproduction *in vitro*. Hence experiments, which perforce ignore many controlling conditions which operate *in vivo*, tend to show maximal reactions under conditions which could not be attained in the human body and on that account ' are of dubious validity without the support of observations in the intact animal '[4].

623. This was stressed by many expert witnesses but would seem to be a fundamental fact ignored or little understood by many of the opponents of fluoridation. The matter is competently explained in the South African Report to which a reference has already been given[4]. A great deal of technical detail is given in the projected Monograph by the World Health Authority (1967)[5] and by Hodge and Smith in ' Fluorine Chemistry '[6] as to the investigations that have been carried out in relation to the action of fluoride on specific enzymatic systems in man, plants and animals. It would be pointless to repeat it.

624. In general, while there would on the results of such experiments appear to be no set pattern, in that fluoride may act to inhibit some enzymes while stimulating others or, at different concentrations, even the same enzyme, the evidence seems to be clear that such mechanisms of

inhibition usually require concentrations of fluoride many times the concentration maintained by the homeostatic mechanisms of the body in the blood plasma[4, 5, 6, 8]. Moreover, nearly all enzymes operate within the intracellular fluids. The evidence that the level of fluoride to be found in those fluids is even lower than that maintained in the plasma has already been referred to (Chapter 9 Sec. (b)).

625. An apparent exception to this statement in the capacity of oral fluoride to block the glycolytic cycle by inhibiting the formation of the enzyme enolase and so preventing the formation of enamel attacking acid, has now been resolved by Hardwick (1961)[9] who has elucidated the fluoride concentrating capacity of the dental plaque. This may range from 6 to 30 ppm; 6 ppm at pH 5.0 being the minimal concentration at which inhibition of the glycolytic cycle has been observed[7].

626. As an illustration of the uncritical use made of *in vitro* experiments in this context I might mention the assertion repeated from more than one source, both lay and scientific[10, 11] that liver lipase was powerfully inhibited by 0.2 ppm fluoride (and stimulated 29% by one part per thousand million). This would appear to derive from a 1906 study[12]. On this study Jenkins[7] (1963) comments:—

> 'These experiments had been carried out in unbuffered media, however, and are valueless. More recent results[13] suggest that lipase is not exceptionally sensitive to fluoride (10 ppm reduces activity to 65 per cent).'.

In other exhibits a similar statement as to lipase being inhibited is attributed to Professor Theorell[14] and other general statements from the same source that fluorine is an 'enzyme poison' have been quoted over and over again. I intend to deal separately with the Swedish experiment of Norrköping (see Chapter 45), as it has been a focal point for criticism of the fluoride hypothesis, but as the views of Professor Theorell, a Nobel Prize winner, command great respect from both proponents and opponents, I think it should be recorded that in 1962 he said—

> 'I stated in my 1958 report that there was a risk involved in increasing the fluorine content of certain organs of the body, and that as fluorine is an enzyme poison, the prolonged addition of fluorine to drinking water might have medical consequences. The Norrköping experiments, however, have not supported this theory. That is something we did not know four years ago. We now know that it is not dangerous.'[15].

627. I have had evidence from Dr Polya of some preliminary experimentation with fluoride in relation to enzymes. As I understood his evidence these were *in vitro* experiments and no results significant to the subject here being discussed were claimed for them. In this regard the witness's main argument in suggesting a halt to fluoridation plans until every known enzyme and every known combination had been fully investigated in relation to different concentrations of fluoride, being outside the realms of practical science, as the witness knew, was based on a social philosophy of resistance to 'compulsion' and not on scientific principles.

628. The same witness also mentioned some experimental work on lipid metabolism of which he was the part author without referring me to the actual results. As the matter was not sought to be pursued I took it no substantial relevance was claimed for it. The subject of fluoride and lipid metabolism is discussed by Venkateswarlu[5]. Other than at levels more than 100 times those attained in water fluoridated at 1 ppm there would seem to be no evidence either epidemiological or experimental implicating fluoride in the aetiology of atherosclerosis or of high serum cholesterol levels. On the other hand, there is evidence that it may have a beneficial role in reducing the incidence of arteriosclerosis (Ch. 43, sec. (c), *post*).

629. But my understanding of the matter is that basically the proof that fluoridation entails no harmful enzymatic effects is clinical and epidemiological. The fact is that in the body a simple physiological effect is generally the summation of numerous cumulative and sometimes alternative reactions catalysed by a number of different enzymes acting either in combination or sequentially at different points along a biochemical route or pathway. This cannot be ignored. To study *in vitro* the reaction of a single enzyme—or even that of a group—or to seek to isolate it in tissue fractions or extracts is like trying to read a symphony from the source of a single instrument. The combinations (not to mention the possible permutations) and the complexing factors are so numerous that I have not been able to understand how in the present state of our knowledge such experiments or biochemical observations made outside the living body could ever afford more than presumptive evidence, and doubtfully that, of what takes place in the living organism. In such circumstances I do not understand the demand that before fluoride is introduced every known enzyme and every known enzyme system should be investigated to the level where an affirmative answer can be given excluding any possibility of harm.

630. It is clear this has not been done, and as far as I can judge in a very technical field, it could never be done. This is not to say that there cannot be and have not been investigations which add significantly to knowledge. The study by Frajola[3] is, in reference to fluoridation, the most advanced work that has been brought to my attention, but because of the limitations of the subject matter which it illustrates better than any determination of a simple isolate it leads to no clear-cut conclusion[4], except that a great deal more work is required fully to describe the nature of the reaction to fluorides at different concentrations of the limited number of systems that he describes. This has to be accepted, and it is for this reason that I suggest that basically, in relation to enzymes and enzyme systems, the safety of fluoridation at 1 ppm must be found in the absence of epidemiological and clinical evidence of harm in the millions who live with it.

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CHAPTER 37. PREGNANCY

631. Fluoride does cross the placenta[1] in all mammalian species examined, including humans[2]. The use of radio-active isotopes would seem to place the matter beyond doubt[3, 4]. The placenta does, however, act as a partial barrier and in the presence of a high maternal intake regulates and limits the amount contributed to the foetal circulation[5, 3]. In such circumstances the foetal blood concentration may be significantly less than that of the maternal blood[3].

632. Fluoride derived by the foetus from the maternal circulation is deposited in the developing bones and teeth. In a low fluoride area the foetal skeletal ash contains about 40 ppm F whereas about 100 ppm F are found in the ash of foetuses from mothers drinking water at 1 ppm[2]. Corresponding differences have been found in foetal teeth[2, 5, 6]. Accumulation is progressive and the rate appears to increase as gestation approaches term[7].

633. There is some uncertainty as to whether fluoride ingested by the mother during pregnancy is effective to inhibit caries in the offspring. An effect on permanent teeth which calcify almost entirely after birth would not be expected and the only report to the contrary by Feltman and Kosel[8] has been discounted[9, 10]. The deciduous teeth on the other hand begin to calcify four to six months prenatally, but the process is not complete at birth and may take another twelve months[2]. Furthermore, the outermost enamel layers are formed last[7]. Therefore, it would seem that the sites of maximum protection and recognisable inhibition are not fully available to fluoridation before birth.

634. It is clear that in relation to all teeth the maximum protective effect is conferred by fluoride taken up by the tooth enamel in the last stages of tooth calcification and this, even in the deciduous teeth, is, generally speaking, a post-natal event. Some recent experimental studies

seeking to demonstrate a cariostatic benefit (which may in this context be a different matter from any general benefit to tooth formation and morphology) from pre-natal fluoride have either failed to show[11] or perhaps have been insufficiently sensitive to show[12] any clear benefit to the foetus.

635. This may yet be found to be due to a quantitative difference in the mechanism of response to fluoride between permanent and deciduous teeth, i.e., the suggestion is that fluoridation at 1 ppm may be insufficient to yield full protection to deciduous teeth which may need a higher concentration estimated at 2 ppm or more[13, 11]. However, as it is the effect on the permanent teeth which is the most important, this suggestion, while affording perhaps an explanation or a partial explanation of the difficulty of demonstrating a benefit due to pre-natal uptake is not an argument for a raised level of fluoridation generally.

636. The matter still awaits final evaluation and there was a conflict of opinion between the witnesses who appeared before the Commission. Professor Storey doubted whether there was any substantial ante-natal benefit and referred to the work of Carlos *et al.*[12] which, however, is not an impressive study as it uses an insensitive index and small numbers[2]. It is, nevertheless, supported generally without the matter being conclusively determined by later work by Katz *et al.*[11], and Horowitz and Heifetz[38]. Professor Martin who was able to refer to unpublished experimental work at present being conducted in his Department[14] was, on the other hand, strongly of the opinion that such a benefit could be postulated though experimental work designed to elucidate the matter is still incomplete. I feel that the balance of authority[2, 7, 15] tends to support the view of Professor Martin. While most calcification of the deciduous teeth occurs after birth the process begins pre-natally and consistently with what is known as to the beneficial effect of fluoride in promoting calcification[39] and in influencing tooth morphology and structure, it would seem reasonable to expect some benefit even if the full effect be masked by post-natal uptake[38]. His view is further supported by studies by Blayney and Hill[16] in the Evanston trials and by Feltman and Kosel in the study already referred to[8]. These studies have been criticised[17] but there is perhaps less reason for doubting the conclusions of Feltman and Kosel in relation to the deciduous teeth than their separate finding in relation to the permanent teeth. Studies by Arnold and his associates in relation to Grand Rapids-Muskegon (previously referred to) tend to the same conclusion[18, 19].

637. The view of Professor Martin that the placenta transmits sufficient fluoride to be of value in the reduction of dental caries to the developing deciduous dentition was adopted after consideration by the National Health and Medical Research Council of Australia as recently as 11 August 1967, i.e., while the Commission was in progress. In its considered conclusion[20], there being ' no evidence that the ingestion of fluoride in normal amounts is harmful either to an expectant mother or to the foetus, the intake of fluoride during pregnancy is recommended '. In coming to this conclusion the Council had its attention drawn to another investigation which had been carried out in Perth, Western Australia, which supported the view that fluoride supplements given to pregnant women are safe and effective in reducing the prevalence of dental caries in the deciduous teeth of children[20].

638. The uncertainties of the matter, however, were reflected in action taken by the Food and Drug Administration, United States Department of Health, Education and Welfare to prohibit the labelling or advertising of pre-natal drug preparations containing fluorides as being beneficial to tooth development in the foetus or in the prevention of dental caries in the offspring[21]. It did so because as stated in the official document—

> ' A number of vitamin-mineral preparations containing fluorides for pre-natal use and intended or represented to be beneficial to tooth development in the . . . offspring have appeared on the market in the last few years.'.

As the administration found at the time there was neither substantial evidence of effectiveness nor a general recognition by qualified experts that such preparations were effective as claimed, it provided by regulation that—

> ' Any such drug preparation that is so labelled, represented, or advertised will be regarded as misbranded '.

639. This action has been misunderstood. It was not intended to raise or suggest any question of safety or undesirable physiological effect[22]. It was intended, as appears plainly from the text, and this has been confirmed by the Department[23] to confine advertising of such preparations to what were regarded as substantiated claims of benefit. Insofar as it is and has been cited as evidencing an official recognition of deleterious results it is misapplied.

640. Other than in relation to mongolism and allergy (two subjects considered separately) no serious attempt has been made before me to establish a case for prejudicial effects in pregnancy as being possible from fluoridation at 1 ppm. There are some suggestions, however, in the earlier

literature which may be thought to have been overlooked if they are not mentioned. Such suggestions seem to stem mainly from the writings of Waldbott and Steyn adopted extensively in the 'propaganda literature', particularly in the more sensational variety such as 'Fluoridation Unmasked' by Fanchon Battelle[24]. They allege uterine bleeding, abortions, stillbirths, drybirth and other obstetric difficulties and foetal abnormalities[25]. All these allegations have been investigated previously by other Commissions of Inquiry. All have been rejected as groundless and some have been exposed as based on untruths or at least a misrepresentation of the facts[26]. The literature and the physiology have been reviewed by many, e.g., Hodge, Smith, and Gedalia[27, 28], separately and in conjunction for the projected World Health Monograph referred to in Chapter 1 (b), *supra*). I find it difficult to perceive the basis on which these allegations rest.

641. Congenital malformations or teratogenic defects are suggested by Steyn[29] as a possible sequel of fluoridation by way of inference from a number of reports implicating drugs and environmental factors none of which are directly related to fluoride. The argument is not direct but the familiar process of broad analogy uncritically applied. The matter was considered in detail by the South African Commission which found that the evidence cited by Steyn was 'fragmentary and unacceptable'. After, nevertheless, reviewing it the Commission concluded—

> 'There is no evidence to support the supposition that fluoride is a teratogenic agent. Extensive studies of the populations of fluoridated areas have shown no increase in the incidence of congenital developmental defects'.

There is absolutely nothing in the wide literature which has been surveyed[31, 32] or the evidence I have heard that would induce me to question this expert conclusion.

642. With regard to the general matter of obstetric difficulties I would refer again to the general review of morbidity and mortality figures contained in Chapter (30) of this Report and particularly to the investigations of the United Kingdom Ministry of Health[33, 34]. I also refer to another Report (1956) by the same Ministry cited and adopted by the New Zealand Commission[26] which, among other matters, compared vital statistics from high- and low-fluoride areas in relation to neonatal and infant mortality and still-birth rates. It found no differences 'between 'high- and low-fluoride areas which could be interpreted as indicating any harmful effect on health, nor even a slight pointer to the need for any further studies to demonstrate this'. I cannot put the matter more effectively than the South African Commission from whose report I again take leave to quote and which I think puts the matter in its proper perspective[35]:—

> '333. The attention of the Commission has been drawn to statements alleging a harmful effect of fluoride on the normal state of pregnancy. It has been suggested, for example, that fluoride causes miscarriages, still-births and neonatal death. These statements have appeared for the most part in the anti-fluoridationist literature and are usually couched in terms which betray an ignorance of the physiology and pathology of the pregnant state.
>
> '334. No increase in the still-birth rate or the neonatal infant mortality rate was noted in Newburgh in New York State compared with the control unfluoridated city of Kingston, and there was no increase in still-birth rate for the area of Sheboygan in the five year period following fluoridation of the water supply.
>
> '335. Exner[36] has referred to the work of Fazekas *et al.*[37], to support his assertion that fluoride is likely to increase the death rate of new-born infants by interfering with their ability to survive conditions of relative anoxia. Reference to the original report of Fazekas *et al.*, reveals that these authors demonstrated that fluoride in doses sufficient to kill a new-born rat in one hour shortened the time that new-born rats were able to survive in an atmosphere of pure nitrogen from fifty minutes to four minutes. This work clearly has no relevance to the question of fluoridation of water supplies and constitutes no grounds whatever for believing that fluoride at a concentration of 1 ppm in the drinking water would adversely affect the infant mortality rate'.

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CHAPTER 38. MONGOLISM

643. Mongolism or Down's Anomaly is a congenital abnormality of human development of a most distressing kind. It is manifested by severe mental retardation and physical dwarfing accompanied by a wide variety of bodily abnormalities. Infantile mortality is high. In fact, before the development of modern antibiotics, it would have been put as high as 50% in the first year of life. Even with their help a 50% mortality can still be expected by the age of 7 and 60% by the age of 20[8, 9]. In 1954 the expectation of life was still estimated at only twelve years in England, ten in Victoria[10, 9]. Acute respiratory infection is the commonest cause of death accounting for 75% of deaths in the first year of life in a Victorian study[9].

644. The suggestion that mongolism is associated with an increased fluoride content of drinking water comes from three papers by Dr Ionel Rapaport published in 1956[1], 1959[2] and 1963[3]. Though he purported to demonstrate a parallelism or correlation only, this has become in the minds of many opponents of fluoridation, equivalent to proof that a causal relationship exists and, quite irresponsibly, it has been represented to the Tasmanian public in press correspondence and other documents as such[4, 5, 6, 7]. I consider the suggestion unfounded for the following reasons.

645. The essential pathological condition has been established only since 1959 as a chromosomal abnormality. Two main types are recognised, the first and by far the commonest, a trisomy in which there is an extra chromosome in one of the diploid pairs of the fertilised cell or zygote, the second a translocation or an abnormal transfer of portion of one chromosome to another. Penrose and Smith[10] describe several mechanisms for the production of the first, the trisomal defect. Their importance to this discussion is that they may indicate different stages in the history of the sex cell to which the pathological event may be related. Cohen *et al.*[11] considers it most likely to occur ' at the first or second meiotic division in the formation of either the ovum or sperm '. In mammals the first stage of meiosis of the gamete in the ovary begins shortly before the birth of the mother or in her early postnatal life[12]. It is completed shortly before ovulation. This does not apply to the sperm cell in which the whole process precedes by a short interval (measured perhaps in weeks) the sperm's passage into the seminal vesicle[13]. Penrose and Smith[10] consistently with Cohen *et al.*[11] would seem to accept the possibility of environmental factors influencing the female gamete at any time during maternal life but do not indicate a view of relative frequency at particular times. The same authors (i.e. Cohen *et al.*) say that ' Less likely, although theoretically possible, the nondisjunction might take place at the gonial stage of the gamete ' which would be shortly before ovulation (up to an interval of weeks) in the case of the female or emission in the case of the male, ' or at the first cleavage division of the fertilised ovum or zygote ' i.e. immediately after conception. Dr Stoller[14] who gave evidence to the Commission considered the ovum to be the vital unit and that the possibility of involvement of the male germ cell would be a very uncommon event. In evidence Dr Stoller[15] favoured the view that the critical time for the majority of cases was related to the second stage of meiosis just prior to ovulation or immediately after conception, though the possibility of an anterior time could not be excluded. This may I think be taken to be the predominant view, applicable at least to the majority of cases.

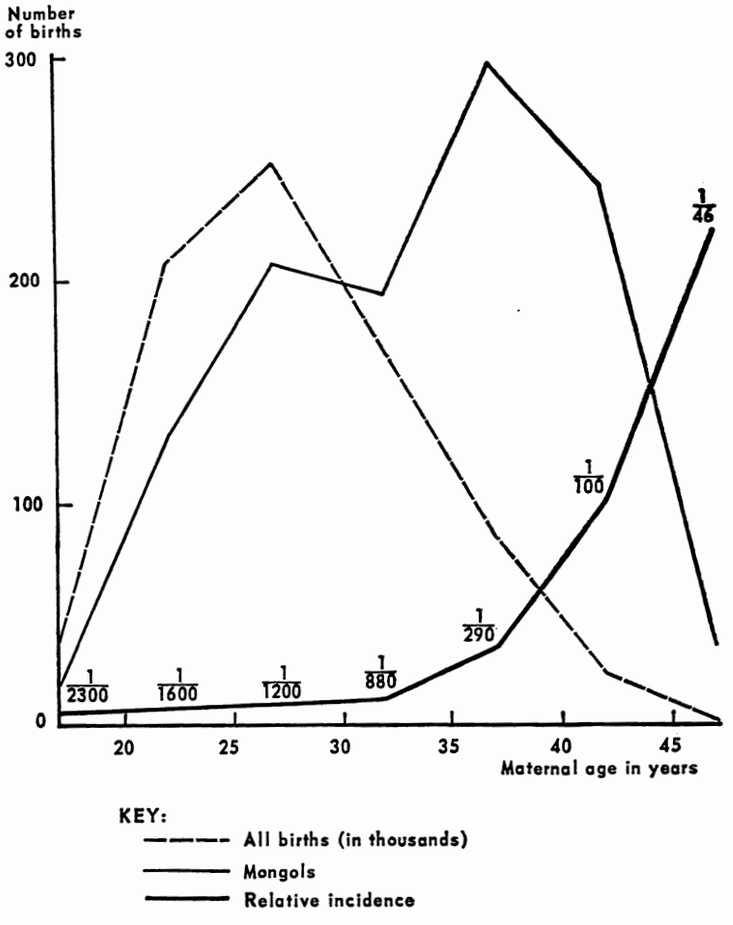
646. The mechanism responsible for the translocation defect is genetically controlled and cannot therefore be related to any definite event in the life history of the parents. The origin must be attributed to the ancestral germ line[10]. However, genetically controlled factors are also known in relation to the trisomy class of defect. Some have a high incidence of transmissibility, e.g. trisomy in the parents causes trisomy inevitably in half the children and, while this condition where the parent is fully affected is rare, a commoner affliction, where a parent is mosaic for trisomy, i.e. exhibiting a variable quality in the affected cells, will still produce the same inevitable result but in a smaller percentage of offspring. In the opinion of Penrose and Smith,[10] these and other genetically transmitted abberrations may be responsible for up to 30% of all cases (though others e.g. Stoller & Baikie would put it lower) but, and this I suggest is the important point in relation to Rapaport's papers. if this be so they must be responsible for a much higher percentage of those born to younger as against older mothers.

647. An important correlation between maternal age and mongolism in the offspring has been recognised for a long time. Incidence in relation to maternal age is shown in Fig. VII.

648. Up to maternal age 30 the incidence rate does not change with age, but beyond that age the relative incidence of mongol births rises sharply. Penrose and Smith[10] have calculated in reviewing the statistics from eleven countries that approximately 60% of all mongol births

FIG. VII

INCIDENCE AT BIRTH FROM SURVEY BY COLLMANN AND STOLLER (1962a)



Taken from Penrose and Smith[10] at p. 151.

may be classified as linked with a maternal age factor and that the mean maternal age for this group would be thirty-eight years, or ten years older than the mean for those other mothers in whom age was not a causative or linked factor. From this the suggestion emerges that there are two types of mongolism, one that is age dependent and one that is not[11]. Support for this has been given by investigations which have demonstrated a preponderance of younger mothers in those cases of mongolism with translocations and other genetically determined defects. Penrose and Smith set out their conclusions in Table XXVIII.

TABLE XXVIII

CLASSIFICATION OF THE DEFINITELY KNOWN AND THEORETICALLY PROBABLE CAUSES OF MONGOLISM IN RELATION TO THE MATERNAL AGE FACTOR WITH ESTIMATES OF THE PROPORTION OF PATIENTS IN EACH AETIOLOGICAL CATEGORY.

Class	*Cause*	*Possible proportion in an unselected group* %
(A) Independent of mother's age	(i) Inevitable non-disjunction (parent partly or wholly trisomic)	10
	(ii) Abnormality in chromosome pairing (inherited translocation, inversion or other anomally of the critical G-chromosome)	10
	(iii) Abnormal meiosis or mitosis (specific genes which cause non-disjunction)	10
	(iv) Environmental disturbance of cell division (infection, poison, radiation)	10
(B) Dependent upon mother's age	Non-disjunction during oogenesis (deterioration of meiotic cell-division mechanism)	60

Taken from Penrose and Smith[10], Table 76.

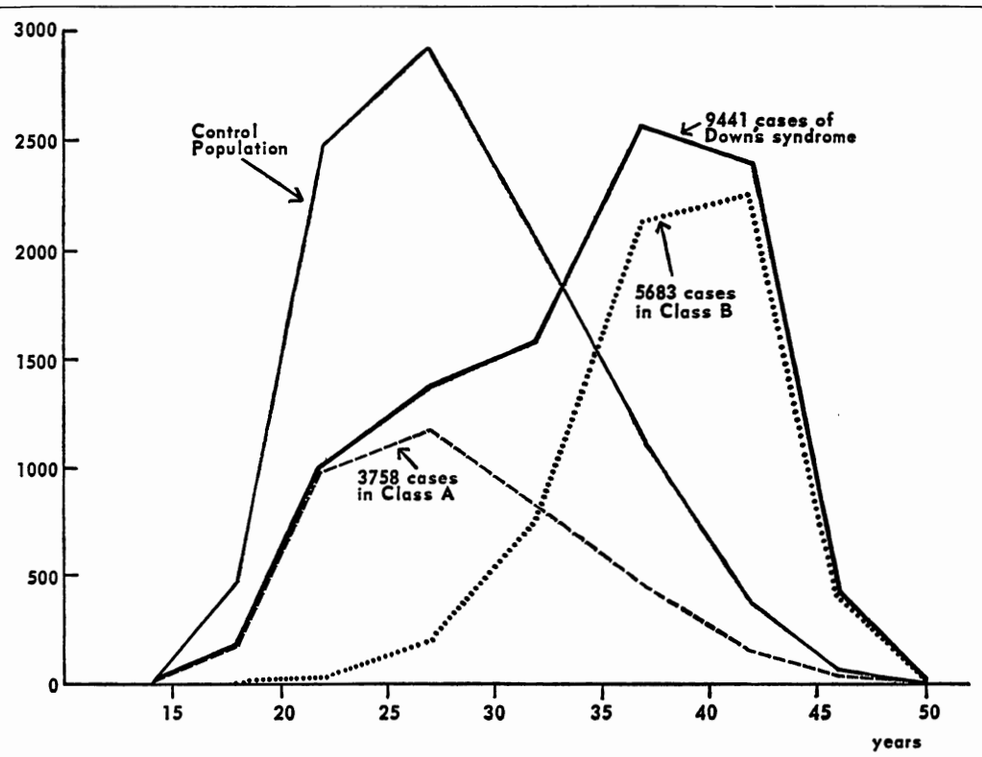
649. In respect of births in Class A—those not linked with maternal age—their incidence will follow the normal maternal age distribution of the population. This is shown in Fig. VIII, again from Penrose and Smith[10] prepared in respect of 9,441 cases of Down's Syndrome from eleven countries.

650. The peak of such births occurs approximately at age 28 but may of course vary by a few years according to the mean maternal age in the country concerned. But there are three times as many births in this group due to inherited factors as there would be to environmental causes (under which head fluoride would fall if it was a cause). A population sample therefore, which because of inadequate sampling methods includes a disproportionate number of Group A cases, and which seeks to attribute an environmental cause to all of them without taking the genetically influenced subgroup (three times as numerous) into account at all, obviously leads to a suspect conclusion. This among other things is what Rapaport would appear to have done as can be seen from his purported correlation with maternal age in his second paper[2].

651. While an environmental cause is postulated by Penrose and Smith[10] for 70% of all cases (but only 10% of Class B) in which no genetically influenced factors are implicated, its nature is unknown, though many have been suggested from alcoholism to contraceptives. In fact it has been said that ' Speculations on the aetiology of mongolism from 1946 to 1959 are so numerous that it is difficult to imagine a factor which has not been incriminated[16] '. It could of course be multiple. What would appear to be a most significant modern study elucidating a possible environmental factor in the causation of this trisomy is the Australian work of Dr Stoller and his colleagues who have demonstrated, in what is regarded as the most thorough epidemiological survey of the subject yet conducted[10, 17], that over twenty-one consecutive years in Victoria there has been a periodicity in the incidence figures with a tendency for geographical as well as chronological clusters. This the authors have interpreted as indicating an infective epidemiology most probably of viral origin[18]. Conformably with their hypothesis they have demonstrated a correlation between epidemics of infectious hepatitis with cyclical peaks of mongoloidism[14, 19, 20, 21].

FIG. VIII

MATERNAL AGE DISTRIBUTION OF 9,441 CASES OF DOWN'S SYNDROME WITH CONTROL POPULATION.



Taken from Penrose and Smith[10].

652. The environmental causes of mongolism not being clinically or pathologically demonstrable, the validity of any epidemiological inferences must rely heavily on the care with which data is gathered. Its prevalence, i.e. the number that may be found in a given community at any one time, can doubtfully only be determined by a survey of the number in institutions unless to these figures can be added the number remaining in private care. Discussion in evidence of the factors that make difficult the collection of reliable data on such a subject, made it obvious

that numbers of institutionalised mongols are of little value as a guide to incidence in the community. Many mongols are not diagnosed at birth even in hospitals,[22] many die in infancy before the diagnosis is established. In relation to birth certificates, except possibly in Scotland, special registers of mongoloid births are not maintained and I am informed that in the United States 'the past and present local reporting of mongoloid births is neither complete nor consistent enough to be statistically reliable'[23]. Witnesses with experience of the matter confirmed that this was the case in other countries as well.

653. Mongolism is not a direct cause of death and even with infections and disorders to which they are known to be predisposed, death certificates may show only the primary cause without reference to mongoloidism as a secondary factor.

654. These difficulties are known and recognised and their importance can be appreciated by a comparison of the extraordinarily detailed methods used by Dr Stoller and his associates in Victoria[18] with those used by Rapaport who included only cases 'of mongolism for which the diagnosis had been put on the birth or death certificates or in the registries of specialised medico-pedagogical institutions'. The differences in the incidence rates reported (Rapaport's total figures being quite plainly out of line with American and world figures) point to the same conclusion.

655. Mongolism is world-wide[10] but careful epidemiological surveys have demonstrated a surprising consistency in incidence figures in all populations of European extraction[11]. Penrose[24] in 1954 found 0.15% as the incidence among populations of European origin as against 0.17 suggested by Oster[25] in 1956 as the average for Europe. Of the fifteen American and European studies reviewed by Cohen *et al.*[11] eleven present estimates of between 1/500 and 1/900 births which disregarding some plainly questionable figures are regarded as 'surprisingly consistent when the geographical and temporal ranges as well as variability in diagnostic criteria and method of ascertainment are considered.'[11] For England and Wales the figure has been estimated to be 0.158 per 100 total births[26] while Collman and Stoller[18] found a figure for Victoria for the same period as taken by the previous authors of 0.157% of live births. This is equivalent to one in 688 live births. Berry in a British study shortly to be referred to which comprised fluoridated as well as non fluoridated towns found an overall incidence of 1/668 births. So closely do reliable modern studies agree in their estimates of incidence that it may be suspected that any study which fails to approach this level of incidence is probably inadequate.

656. Rapaport published his first paper[1] in 1956. Even allowing for the fact that the chromosomal abnormalities which are now known to be pathognomonic had not then been described, the study was inadequate in design, inefficient in execution and anomalous in results. Though he claimed that a statistical study of the geographical distribution 'brings to light a parallelism between the prevalence of this affliction and the fluoride content of drinking water' in the next sentence he says—

> 'An artificially-produced increase in the fluoride content of water, practised over the past five years in Wisconsin and for two years in Illinois, does not seem to increase the prevalence of mongolism in any noticeable way.'.

This passage seems generally to have been overlooked. However, of this paper I think it is sufficient to say that nobody, and I include at least the responsible opponents of fluoridation, now purports to rely on it.

657. Its main faults were that he used actual numbers of mongol births without relating them to the number of live or total births in the community; that even with those it could not be ascertained from the data and records he consulted that his sampling of the mongol population was adequate; that he made his correlation with the place of birth which may have been an urban hospital and not with the place of residence of the mother at the time of conception—in fact corrected figures disclosed a residence error as high as 45.7%—and that his water histories were in any case inadequate.

658. Its shortcomings as a study were pointed out to him at great length by Dr A. L. Russell, Chief of the Epidemiology and Biometry Branch, National Institute of Dental Research, United States of America. This letter is too long to summarise but its importance in the history of this matter is so great that—having first referred it to Dr Russell for verification—I reproduce it as an appendix, with another letter (also referred) written by Dr Russell to the Senior Dental Officer of the English Ministry of Health, United Kingdom, bearing on the same matter (Appendices 'I' and 'J').

659. In 1959 Rapaport published his second paper in which he said that:—

> 'Following the advice of Dr A. L. Russell, Chief of the Department of Epidemiology and Biometry of the National Institute of Dental Research in the United States, we have limited our investigation to a single State, Iillinois. The Department of Public Health of this State has communicated to us the chemical analyses of the potable water of all the towns with 10,000 to 100,000 inhabitants.'.

As to this claim, Dr Russell's comments[30] can be left to speak for themselves:—

> '. . . All I know about the 1959 report can be summarized as follows:
>
> 'I first heard about this paper in March of 1960 when Dr John E. Bowers, Dean of the medical school of the University of Wisconsin, telephoned me on behalf of his faculty board to inquire if it was true that I had worked with Rapaport in the study, had reviewed his evidence, agreed with his conclusions, and had authorized the use of my name in the report. When Dr Bowers learned that I had never seen the evidence or the manuscript, he sent me a copy and directed Rapaport to report to me in my office here, with his raw data on March 28 1960. He (Dr Bowers) asked me to inform him of my opinions immediately, by telephone, as some sort of faculty action was imminent. Rapaport did not appear at the time set, and I have not heard from him since. Since it would evidently have been very useful to him if he had been able to convince me that his work was valid, I can only infer from his failure to try that he knew of flaws in the work grave enough to nullify his conclusions.
>
> 'In a situation like his where overall differences were so small, little bias is necessary to influence the conclusions. The misclassification of only two cases per year over the term of seven years would be enough to account for the whole of the difference. Our birth records are notoriously incomplete and misleading for information of this character. There is no reason to believe mongolism is any less prevalent in Illinois than elsewhere in the United States or the world. Competent observers using direct evidence have reported the frequency of mongol births to be as high as one in 600 live births, and rarely lower than one in 700.
>
> 'If we are to accept Rapaport's data at face value the State of Illinois (fluoride and all) produces only one mongol for each 2,250 live births. I believe that the writer of *The Scope of Epidemiology in Psychiatry* (In the W.H.O. Chronicle for January, 1963) would agree that the entire paper is worthless.
>
> 'We had concluded, before this episode, that Rapaport was hopelessly inept. His performance here led me to doubt his integrity as well. Despite my obvious prejudice, I hope this information will be useful to you.'.

660. The question is what did Rapaport do to correct his first study and remove the criticism that previously attached. It does not really appear. His figures now appear as relative incidence figures, i.e., as related to 100,000 live births and all they disclose is an incidence equivalent to approximately one-third only of the American and world figures. Information supplied by the Division of Dental Health, Department of Health, Education and Welfare of the United States suggests that he made no effort to revise the water histories previously criticised as inadequate[23] and a purported correlation with maternal age is so anomalously expressed that I have difficulty both in following the argument or accepting the inferences suggested. Attempts by Counsel assisting the Commission to get in touch with Dr Rapaport to get him to comment have not produced any reply but there is a suggestion that he is unavailable[28]. It appears that he is no longer a member of the University Faculty to which he refers in his work. It also appears that the Faculty has disassociated itself from the work and the conclusions drawn[29].

661. A later paper[3] published in 1963 repeats the 1959 figures without addition or explanation. It adds data concerning the caries rate in institutionalised mongols and some other data concerning the influence of fluoride on melanoma formation in fruit flies. Witnesses before me have described such matters as naive and irrelevant in their context but whether they would be in a properly established inquiry is perhaps uncertain. In relation to the teeth in particular there are many reports of dental and gum abnormalities in mongols which reflect the general constitutional disturbance[10], but the failure of the paper to attempt to account for other factors that may have been conducive to the caries rates observed is what was mainly criticised. What does seem clear is that no reliance at all can be placed on a correlation demonstrated in such suspect circumstances, and I note that no body of inquiry has been prepared to do so[31, 32, 33, 34].

662. As a speculation, however, the implications of Rapaport's suggested correlation are serious, and they have been so regarded. In 1958 a study[27] was mounted in England to see whether

any confirmation of his theories could be found. A much more thorough survey of mongol incidence was made over ten years in the nine cities and one county area selected for the study and the reliability of the figures so obtained is attested both by the methods used and the comparability of the results obtained with established incidence figures. No confirmation of Rapaport's speculations was obtained and in fact the conclusion of the author was unequivocably ' No indication emerges of any relationship between the level of fluoride in the water supply and the incidence of mongolism '[27].

663. The Division of Dental Health of the Department of Health, Education and Welfare of the United States has made the following comment[23]:—

> ' An approximation of the rate of mongoloid births was recently reported to the U.S. Public Health Service by 29 States for the years 1962, 1963 and 1964*. When these figures are compared with the proportion of the State population served fluoridated water, no consistent correlation between these factors can be detected. For instance, of the 29 States providing data on the rates of mongoloid births to the number of live births, the State reporting the highest mongoloid birth rate in 1962 was one of the lowest in percentage of population supplied with fluoridated water; the second highest mongoloid birth rate was in the State with a high proportion of State population served fluoridated water. The third highest mongoloid birth rate (only one percentage point lower) was reported from the State that ranks lowest in the proportion of population supplied with fluoridated water. A similar inconsistent relationship between these two factors was observed among the other States confirming the absence of any relationship between the frequency of mongolism and the consumption of fluoridated water.
>
> * Based upon a 1% sampling.'.

664. I was able to discuss this matter and Rapaport's papers with two witnesses who may claim to special knowledge of the subject both in relation to its causation and its epidemiology. They were Dr Stoller whose work has already been referred to and Professor Baikie who has been associated with research into the correlation between leukaemia and mongolism. Neither were prepared to accept Rapaport's methods or his conclusions. I can only report that on the evidence these speculations do not constitute a reason for inferring a causative role for fluoridation at 1 ppm in the aetiology of mongolism.

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CHAPTER 39. ECTOPIC CALCIFICATION

665. The argument has been raised from time to time (sometimes by way of crude analogy based on the action of fluoride in promoting the formation of bone; sometimes by way of deduction from some early and imperfect observations in which high fluoride concentrations have been noted in calcified deposits) that fluoridated water may promote ectopic calcification within the human body resulting in such things as calcified arteries, kidney and bladder stones and other similar pathological conditions. The argument is supported generally by Steyn[1] and Waldbott[2] in particular. It is also supported by reference to Alfred Taylor[3] and his experiments with mice which have been discussed in Chapter 32 (a), *supra.*

666. There is quite an extensive literature on the theoretical and physiological aspects of the matter. This is reviewed in the South African Report[4]. There is of course a fundamental distinction between the biological formation of bone as a living organic substance and ectopic calcified deposits which are not a function of cellular or osteoblastic activity. However it seems clear that when ectopic calcification does occur the bony salts which are deposited in the form of hydroxyapatite will bind fluoride taken from the body fluids as does the apatite in bone and teeth. Such deposits are not normally subject to resorption and with crystal ageing the fluoride so taken up is inactivated in the calcified deposit.

667. The overwhelming weight of authority supports the view that ectopic calcification as a pathological process is not in any way induced by dietary fluoride. Its presence in such deposits is a secondary phenomenon which can be correlated not with the degree of fluoride exposure or ingestion but with the degree of calcification present. It is completely explicable on physiological and chemical grounds and is in no way detrimental to health[5, 6, 7, 8, 9, 10]. The point is graphically made by one of the authors referred to: 'Thus to attribute stone formation to fluoride is hardly less far-fetched than to say that ink causes blotters.'.

668. Epidemiological studies support this view. They do not indicate any differences in the incidence of kidney or urinary calcification between high- and low-fluoride areas. Indeed the suggestion emerges (later commented on under the heading of adult benefit (Chapter 43, *post*) that fluoride by inhibiting resorption of bone in osteoporotic states may act beneficially to retard or prevent such potentially serious matters as aortic calcification[11, 12]. There is some experimental evidence in relation to kidney stones in rats which supports a similar inference[13].

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CHAPTER 40. FLUORIDE AS A 'CALCIUM ROBBER'—FLUORIDE AND CALCIUM METABOLISM

669. If fluoride was to the opponents of fluoridation the arch villain of the drama, then calcium was the shining white knight. Unfortunately many of the statements and arguments lacked scientific backing and seemed to be based more on the intuitive reasoning of folk medicine than biochemical knowledge. Their attitude is epitomised by Waldbott[1]—'Fluorine is known to attach itself to calcium wherever it can find it in the system'. For this he cites Roholm, but I have been unable to verify the reference.

670. As a preliminary observation I note that differences in serum calcium levels in cases of high fluoride intake have not been demonstrated. Roholm's determination of the serum calcium of eighteen cryolite workers with skeletal fluorosis led him to remark of the results 'undoubtedly they may be regarded as normal'[2]. This is consistent with the negative findings on the same matter by Leone *et al.* (1954) in the case of the Bartlett-Cameron study[3] and by Bernstein and Cohen[4] in patients given large doses of sodium fluoride for the treatment of osteoporosis.

671. As the normal serum concentration of calcium is 100 ppm of which at least half is free ion and the serum concentration of fluoride is below 0.2 ppm, the remarkable thing is, if this suggestion were true, that any fluoride ever reaches bone or teeth at all. But it does and it does so not in the form of insoluble calcium fluoride which indeed is not formed within the body, but as free ion which exchanges for carbonate and hydroxyl ions—not it should be noted for calcium ions[5] (Ch. 9 (d) and (e), *supra*).

672. Calcium is another abundant element. It is a normal and an important constituent of diet. For Australians the present daily dietary intake of calcium for healthy persons, normally vigorous, living in a warm temperate climate recommended by the Nutrition Committee of the National Health and Medical Research Council[6] is from 400 to 800 mg a day. For children the range is from 400-1,400 depending on age, the maximum (600-1,400) being for boys in the age group 11 to 15.

673. In the United Kingdom the average intake has been calculated at 1,204 mg per day and the figures for the United States would seem at least to be comparable, if not higher[7]. This should be compared with a calculated intake for all ages by the World Health Authority for India and Japan of 469 and 384 mg respectively[7].

674. I have no figures for Australian intakes though no reason has been suggested why they should differ significantly from those given for the United Kingdom and the United States. Professor Collins gave general evidence to this effect.

675. However, for Tasmania the picture may display some differences. Mrs Coy, Nutrition Officer of the Department of Health Services, gave evidence of a dietary survey in 1944 conducted by the National Health and Medical Research Council in respect of 70 metropolitan and 134 country families which revealed a dietary standard lower than the rest of Australia, particularly in relation to calcium intake. Later information, except as to children, is not available. As to them, surveys made between 1954[8] and 1959[9] of limited numbers of primary and high school children revealed that 66% of 169 high school children failed to reach the standards for adequacy and 30% did not attain 50% of the required amount, i.e., 700 mg, due, in the opinion of the investigator, to a too small milk intake. The figures for the primary school group were better but still substantially below the recommended level. However, in this survey the figure of 1,200 to 1,600 mg seems to have been taken as the required daily intake[9] and these figures may need revision. Nevertheless they undoubtedly reflected a disappointingly low standard. Efforts have since been made by the School Health Service to correct this but with what quantitative results is not known to me, though the evidence is that it is improving 'considerably'. This low intake is all the more striking because a pint of whole milk a day alone would give 715 mg of calcium, while 7 oz, which is the school free ration, or 1 oz of cheese will give approximately 238 mg.

676. On the basis of the United Kingdom and the United States figures, one could conservatively estimate that the average Australian diet might contain at least 1,000 mg of calcium a day. In fact, 400 mg will meet the needs of the great majority of people[6] and with children with intakes as low as 200 mg a day no clear-cut effects of a lack have been shown[10] so that the margin is large. Excretory losses have to be allowed for but the absorption studies listed by Bronner[11] show rates ranging from 25% to 70% in subjects at varying age from 11-72. The last figure is probably atypical having been obtained in a 72-year-old man previously on a low calcium diet. For an adult man on an intake of 1,000 mg a day the amount of dietary calcium absorbed is typically taken as 300 mg or 30%[11].

677. So far only calcium from food sources has been considered. Calcium salts are also found in drinking water and some free calcium ion. A soft drinking water may have up to 50 ppm of hardness expressed as calcium carbonate and hard waters even greater amounts. The maximum allowable by World Health Standards for potable water is 200 ppm[12] but the maximum 'acceptable' limit is regarded as 75 ppm (*ibid*). Drinking water is not normally regarded as an important source of dietary calcium in view of the normally large margin available from other dietary elements. However, in time of rationing in London, it was calculated that an average intake from this source was approximately 110 mg daily[13].

678. It is necessary to set these matters out so that some facets of this argument can be seen in a perspective that a layman can appreciate. A molecule of calcium fluoride contains one atom of calcium and two of fluoride. However, because of their different molecular weights, the molecule would contain 51.33% calcium and 48.67% fluoride so that if one could postulate that every mg of fluoride drunk combined with calcium, the maximum calcium so absorbed by each mg of fluoride would be approximately 1 mg. In other words an intake of 3 mg of fluoride a day, could serve to remove a maximum of 3 mg of calcium in a daily intake of approximately 1,000.

679. But, of course, this does not happen. It is agreed that calcium salts can complex with fluoride in the gut and diminish absorption of fluoride. In such a case it is assumed that some insoluble or poorly absorbable compound is formed and excreted via the bowel. For this to happen the presence of free ions is necessary in such concentrations and under such conditions of pH as to favour precipitation. This is not a haphazard process but one to which there are mathematical limits expressed in terms of solubility products and precipitation factors.

680. The evidence of Professor O'Donnell, Professer of Inorganic Chemistry, University of Melbourne, who is, incidentally a specialist in fluorine chemistry, gave a value of 2×10^{-10} to the solubility product of calcium fluoride. This would mean that in water containing 1 ppm of fluoride and with free ions of calcium present, theoretically precipitation would not occur under a concentration of 3,200 ppm of calcium.

681. I felt that Professor O'Donnell spoke with more authority on this matter than any other witness. However, another solubility product was suggested of 3×10^{-11} and, just as a point of interest, the same calculation, using this value, would still require a calcium concentration of 480 ppm. Taking even this value and assuming water containing 500 ppm of calcium (approximately fifty times greater than most Tasmanian waters) and equating 'real' precipitation with incipient precipitation, the nett result would be that even this concentration of calcium would be sufficient only to precipitate 2% of the fluoride. If, in the same calculation, a calcium figure of 1,000 ppm were used with the same value of the solubility product constant, he has calculated that over 70% of the fluoride would remain in solution even if all possible precipitation went to completion.

682. At my request he has supplied a table (Table XXIX), utilising both values suggested for the solubility product constant and showing the values of calcium required for incipient precipitation at different levels of fluoride.

TABLE XXIX

LEVEL OF CALCIUM (PPM) REQUIRED FOR INCIPIENT PRECIPITATION

Solubility Product	*Fluorine Levels*		
	1 ppm	5 ppm	10 ppm
$K_s = 2 \times 10^{-10}$ (O'Donnell)	3200	128	32
$K_s = 3 \times 10^{-11}$	480	19	4.8

683. The precipitation factor may of course be varied by increasing the fluoride concentration. However, as the solubility of calcium fluoride is 16 ppm in any given fluid medium it would need 8 ppm of fluoride before the solubility factor would be exceeded and precipitation could be expected. This may be affected by a lower pH but in the direction of raising the solubility not lowering it. Thus Feltman and Kosel found in their study of the administration of calcium fluoride tablets that in gastric juice (pH 2.5) the calcium fluoride was 50 ppm soluble[14].

684. In the light of these basic facts so many of the arguments about calcium and fluoride become meaningless. There are two situations, one relating to water and the other to food. Dealing with drinking water and the question of any effect on ingestion, it seems plain that no difference in the calcium or fluoride available for absorption, either quantitative or qualitative, can within practical limits, be related to the content of either ion in the water.

685. Food raises other questions. It is generally accepted that calcium in the diet can diminish fluoride absorption (it is the customary antidote for toxic doses) though the evidence is confusing. Probably insufficient attention has been given to the necessity for simultaneous ingestion of both fluoride and the neutralising food[14]. Thus owing to the speed with which fluoride is absorbed calcium phosphate taken fifteen minutes after ingestion of an aqueous solution of fluoride, had a much diminished effect in lessening absorption[15]. Though the precise manner in which the reduction is effected is unknown it presumably reflects dietary concentrations of calcium and calcium salts quantitatively and qualitatively different from those in solution in drinking water[16]. Milk, for instance, contains approximately 1230 mg of calcium per litre[17] and calcium phosphate as found in bone salts is able to bind fluoride even from low concentrations[18].

686. It is a matter of diminishing, not removal. Thus rats fed fluoride in a 1% calcium solution (10,000 ppm) absorbed 24.8% of the fluoride as against a control group fed the same amount in distilled water which absorbed 89.5%[19] and even in milk the amount of fluoride which will be taken up is very small before the interfering effect of the calcium will be overcome[20, 16]. But the point of all this is to emphasise that proportionately to intake, it is the fluoride that suffers, not the calcium.

687. Once absorbed from the diet the homeostatic forces which regulate the serum concentration of both calcium and fluoride operate to prevent any interference with the normal physiological metabolism of both[16]. In fact, calcium fluoride is not formed in the human body[11, 21] (excepting of course in the gut before absorption) nor has it ever been found in the bones or in the teeth of the skeleton[25]. It has been calculated that it could form where the serum concentration of fluoride equalled or exceeded 100 ppm[22]. In terms of human physiology this is an impossible level. It, calcium fluoride, has been demonstrated as a temporary precipitate on the surface of teeth after topical application of a very strong solution (1% of fluoride i.e. 10,000 ppm)[23, 24, 32] but this is of no physiological significance. Hence I think I should report categorically that I have found no physiological basis (except at blood serum levels which would be toxic in themselves) for statements such as the following if meant to be related to the body fluids and to conditions within the living body:—

> 'However, once fluorine (fluoride) enters the system it has a strong tendency to combine with calcium thus de-ionising it and rendering it unavailable for many and very important physiological processes in the body;'[26]

and

> 'Fluorides are anticoagulants. This probably results from the precipitation of calcium . . .'[27].

No evidence was produced to support a related suggestion that fluoride was connected with haemophilia which is a genetically determined condition. Roholm considered it but found no evidence to support it[28] and the suggestion has been dismissed by every authority that has considered it[29].

688. The action of fluoride on bone substance and the teeth has already been referred to (Ch. 9, *supra*). The process described by Neuman *et al.* (1950)[18] whereby the fluoride ion exchanges with hydroxyl and bicarbonate groups in the apatite lattice does not involve any competition with calcium or phosphate, the content of which is not diminished[30, 31, 32]. In fact so far from 'robbing' bone salts of calcium the effects of fluoride is to stimulate its deposition and to enhance calcification[10, 33]. This is the basis of the therapy previously mentioned in treating disorders of bone calcification and of the strongly evidenced suggestion that fluoride actually acts to prevent the osteoporotic disabilities of middle age. Among other fluoride effects noted in these conditions is that of modifying and lessening negative calcium balance, i.e. under the action of fluoride the body retains more calcium than formerly[34, 35] (see Chapter 43 (b), *post*). This has been confirmed in experimental animals[30].

689. In the rat, Singer *et al.*[36] have shown that drinking water fluoridated at 50 ppm had no effect on the metabolism of dietary calcium as far as it related to the bones and teeth nor was the retention and turnover of the calcium originally present in the calcified tissue in any way affected. The further role to which Professor Storey has drawn attention and which suggests how fluoride may regulate the calcium phosphorus ratio of the plasma is to the benefit of calcification, not the reverse[37].

690. The osteoporosis which has been described in advanced cases of chronic fluoride toxicity (bordering on the subacute) is a complex process usually accompanied by production of exostotic bone[38], i.e. both excessive bone formation and resorption due to toxic interference with osteoblastic and osteoclastic systems are products of the same mechanism and reflect the same general level of toxic dosage[39]. As physiological processes they have no relevance to fluoridation at 1 ppm.

691. The conclusion therefore is that fluoridation will not initiate physiological processes that will rob the body of calcium. On the contrary, at the levels proposed the effect will be to enhance calcification and to promote better bone growth, both in the young and the aged[10]. If anything, a high calcium diet may 'rob' the body of fluoride but the reverse is not chemically sustainable[29, 40].

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CHAPTER 41. WHETHER THE EFFECTS OF FLUORIDE CAN BE RELATED TO THE HARDNESS OR SOFTNESS OF THE DRINKING WATER

692. Linked with the matters discussed in the preceding chapter is the suggestion[1, 2] that the effects of fluoride in drinking water may be substantially mitigated by the hardness of the water, and that in the absence of any such protective effect there is risk of excessive dental fluorosis and possibly other side effects if fluoride is introduced into soft waters. More specifically it is put that hard waters contain, by reason of their calcium content, a natural detoxifying agent which combining with the fluoride to form calcium fluoride permits it to be excreted harmlessly from the body.

693. Let it be admitted at once that Tasmanian waters are soft, very soft, particularly in Hobart and Launceston. Dr Sutton[1] quoted the following official figures:—

	Ca ppm	Mg ppm	Total parts hardness
Hobart—			
Reservoir 1	5–6	2 –3	18–27
Reservoir 2	2–6	0.5–0.7	9–30
Reservoir 3	5–7.5	2 –3	17–30
Launceston	5	1.7	30

694. Total hardness may include carbonates as well but no suggestions were made respecting such parts of hardness, perhaps for the reason that as the South African Report puts it[3]—

> 'The carbonate fraction of the enamel is variable and unevenly distributed in the enamel. It may have some bearing on caries susceptibility because its presence inhibits the proper crystalisation of apatites and increases the rate of solution of the apatite in acids.'.

It is not, therefore, a very helpful factor in any argument suggesting that hard water may, as it were, protect teeth from the effects of fluoride. In any case, for chemical reasons, the carbonates whether of calcium or magnesium are not important as competitors in this matter of the interrelationship of calcium, magnesium and fluoride[4].

695. Some matters as to calcium and its power to complex fluoride, either in the drinking water, the gut or in the body fluids, have already been mentioned and discussed in the preceding chapter. At physiological concentrations its power to complex fluoride is negligible. Water fluoridated at 1 ppm would need free calcium ion at a concentration of approximately 2,800 ppm in the same liquid for even a small percentage of it to be precipitated, and while it may not be strictly accurate to extrapolate directly from this to its effect in the body fluids it has been calculated that no complexing of fluoride and calcium is likely under 100 ppm of fluoride[5]. Physiologically this is an impossible level and calcium fluoride has never been demonstrated in the living body.

696. There may be more significance in magnesium though it was barely mentioned in argument. The World Health Organisation recommends[6] 50 ppm as the maximum acceptable concentration in potable waters and 150 ppm as the maximum allowable. These are high values. For the purposes of the Commission and to facilitate the discussion of this particular matter, Professor Storey has tabulated the calcium and magnesium values in the drinking water of the thirty-eight American cities studied by Dean and his associates in the various papers of the monograph 'Fluorine Drinking Waters'[7] on which this argument is based. In sixteen out of the thirty-eight studied, the magnesium value was less than 10 ppm. In seven only did it exceed 40 ppm and the highest value was 72.6. Table XXX gives details. In its preparation, for convenience, the places named have been grouped in ascending order of fluoride content and they have been classed into four groups as appears in the table.

TABLE XXX

	F	Ca	Mg	Fluorosis Index
FLUORIDE CONCENTRATION 0.4–1.3—				
1. Elgin	0.4	14.9	15.9	0.22
2. Pueblo	0.6	83.6	27.3	0.18
3. Big Springs, Tex.	1.1	73.2	6.6	0.08
4. Junction City, Kansas	0.7	83.5	17.9	0.03
5. Mullins, Sth Cal.	1.0	1.3	0.8	0.23
6. Aurora	1.2	88.5	28.0	0.32
7. Joliet	1.3	89.2	30.8	0.46
FLUORIDE CONCENTRATION 1.4–2.1—				
8. East Moline, III	1.4	54.3	25.7	0.49
9. Howard	1.4	215.5	57.7	0.57
10. Maywood	1.4	20.0	6.1	0.30
11. Webster City, Iowa	1.5	105.5	50.5	0.35
12. Aberdeen (Wash. St.)	1.6	217.2	34.1	0.48
13. Monmouth	1.8	69.3	26.6	0.58
14. Aberdeen (W. Hill)	1.8	240.0	53.3	0.48
15. Galesburg, Ill.	1.9	62.2	25.7	0.69
16. Elmhurst	1.8	86.9	25.9	0.67
17. Iroquois	2.1	11.4	3.5	0.86
FLUORIDE CONCENTRATION 2.2–2.8—				
18. Clovis	2.2	25.7	21.8	1.37
19. Miller	2.2	185.8	52.4	0.83
20. Leola	2.3	34.3	3.5	0.80
21. Wolsey	2.4	140.0	32.8	1.54
22. Gettysburg	2.4	14.3	0.9	0.98
23. Colorado Springs	2.6	8.0	1.6	1.3
24. Carthage	2.6	41.4	15.3	0.92
25. Onida	2.6	111.5	9.6	0.65
26. Redfield	2.7	40.0	4.4	1.01
27. Ipswich	2.8	323.0	65.5	1.26
FLUORIDE CONCENTRATION 2.9—				
28. Plainview, Tex.	2.9	48.5	35.8	1.8
29. St Lawrence	2.9	132.2	38.4	1.8
30. Doland	2.9	17.2	0.9	1.52
31. Platte	3.0	22.9	8.7	1.42
32. Conway, S. Car.	4.0	2.3	0.5	2.1
33. Lubbock, Tex.	4.4	60.7	72.6	2.66
34. Pierpoint	4.7	14.3	5.2	1.93
35. Hecla	5.0	54.3	5.2	2.6
36. Amarillo, Tex.	5.9	43.9	47.8	2.2
37. Lake Preston	5.9	14.3	4.8	2.4
38. Britton	6.6	25.7	3.5	2.3

697. These figures can be set alongside a definitive study by Feldman *et al.*[4] in which they calculated the complexing power of various metallic ions in solution in water fluoridated by the addition of sodium silicofluoride to yield a concentration of 1 ppm F. Taking a list of metallic ions which may be found in water supplies, they calculated the lowest concentration of each that would complex 1% of the total F ion. Comparing the quantities so found with the maximal concentrations established by others[8] as typical of water supplies found in the United States, they concluded that 'the only ones possibly present in sufficient concentration to complex at least 1 per cent of the fluoride are calcium, magnesium, tin, chromium, aluminium, iron, and titanium' but that of these 'only calcium and magnesium actually complex more than 1 per cent of the F^-'.

698. As to calcium and magnesium, they express their conclusions in the following form:—

DISTRIBUTION OF F IN DRINKING WATER

Sample	pH	Total Ca (ppm)	Total Mg (ppm)	% Total F as			
				HF	F^-	CaF^+	MgF^+
A	5	1	1	1.5	98.2	0.03	0.3
B	8	1	1	0	99.7	0.03	0.3
C	5	100	100	1.5	67.7	2.8	28.0
D	8	100	100	0	69.2	2.8	28.0

The usual pH values of drinking water are between 6 and 8. At pH 8 100 parts of Ca would complex 2.8% of the fluoride ion while 100 ppm of magnesium (a very high figure indeed) would complex 28% only. But the complex thus formed CaF^+ is a soluble entity and remains with solution. It is not to be confused with the species CaF_2 which is sparingly soluble and for which precipitation factors have been discussed in the preceding chapter (9).

699. As Professor O'Donnell concluded in a communication to the Commission[10] chemically there would seem to be no practical situation in which complexing or precipitation of fluoride from fluoridated water by other ions present in the same water would be of any significance whatever.

700. Perhaps I should refer briefly here to a matter raised in evidence as to the efficacy of some pills containing 2 mg of calcium, 2 mg of magnesium and 2 mg of phosphate, compounded by a Hobart pharmacist to a formula supplied by a witness before the Commission, designed to 'detoxify' a person taking them against the effects of fluoridation at 1 ppm. The witness plainly did not believe in the practical efficacy of the formula supplied nor do I. The matters already discussed and the relevant passages in the transcript[11] sufficiently indicate my reasons.

701. However, the argument as to hard water containing some protective factors was raised mainly in epidemiological form. It has its genesis in a remark made by Deatherage in 1942[12] in which, referring to some naturally occurring soft fluoride-bearing waters, he says—

> 'It is these soft waters which cause the most severe mottled enamel.'.

He gives no data and the only elaboration is contained in a subsequent passage—

> 'Factors other than the fluoride content of water have some influence on the degree of mottled enamel resulting from its use. One of these is the amount of water actually consumed, and this in turn depends to a large extent on the mineral content and the palatability of the water. Of two waters with the same fluoride content, the one with the normal mineral content of 350 ppm showed an incidence more than twice that of the other water with a mineral content of 1,900 ppm.'.

He does not identify the two waters to which he is referring—indeed no figures for hardness are contained in the article—but this observation has echoed down this controversy over the years.

702. I would agree with Professor Martin[13] that it is impossible to identify from Table 1 of the same article the two waters referred to as belonging to the towns of Geneva and Kewanee. Kewanee was studied by Dean[14]. He gives the full water analysis and his residue on evaporation of 1,908 ppm agrees approximately with Deatherage's rounded figure for 'mineral content' of 1,900 ppm. It is doubtful indeed whether it could be called potable water having calcium 115.7 ppm, magnesium 38, sodium and potassium calculated as sodium 506.2, bicarbonate 300.1, sulphate 308.6, nitrate 6.6, and chloride 689.0 ppm[14]. When one takes all these into account one wonders just how much would be drunk or even used for cooking. As to Geneva, I have no details but with a mineral content of 350 ppm, it is not obviously soft. Though Deatherage himself did not

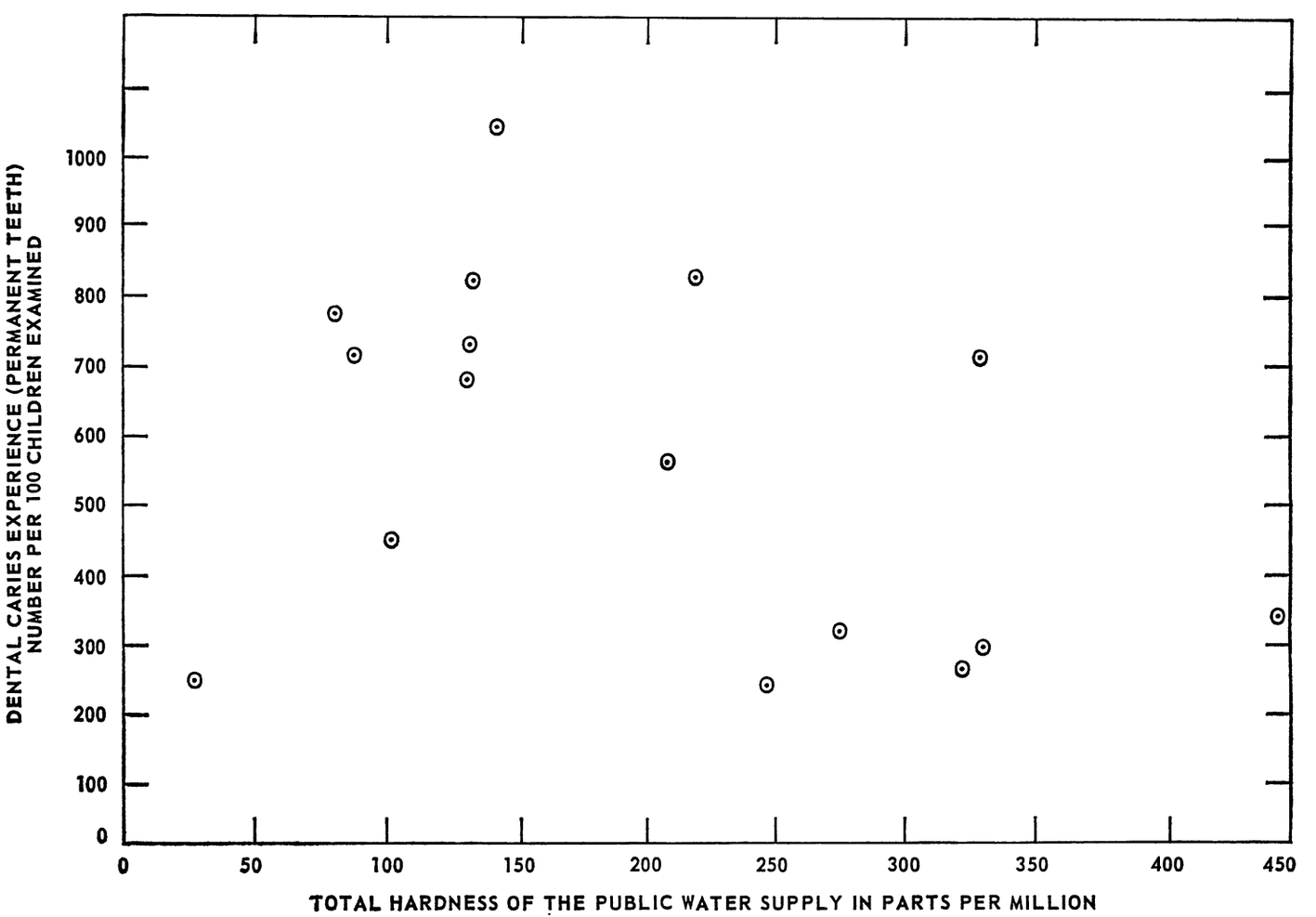
make the calculation, the computed fluorosis index from his data for the two places would be .4 for Geneva and .18 for Kewanee, both below the level of public health significance[15]. However, the only 'mild' cases recorded for both places are all in Kewanee (2.1%) as against none for Geneva. On such a slender basis does this argument apparently depend.

703. Dean[18] himself rejects the suggestion. He examined the matter at some length and referred to previous studies which previously to the discovery of the role of fluoride had found an association between hard water and freedom from caries. His own findings, he considered, indicated that the observed differences in dental caries experience were not explicable on the basis of water hardness. His plot (Fig. IX) which I reproduce hereunder showed a random distribution with no statistical trend or correlation between caries reduction and hardness. As he said:—

> '. . . wide quantitative variation in another factor (hardness) may occur without noticeably influencing the phenomenon, suggestive of an association without causal connection.'.

FIG. IX

VARIATION IN DENTAL CARIES EXPERIENCE RATES OBSERVED IN 7,257 SELECTED WHITE SCHOOL CHILDREN, AGED 12-14 YEARS, IN 21 CITIES OF 4 STATES WITH THE TOTAL HARDNESS OF PUBLIC WATER SUPPLY



Taken from Dean (1946)[18].

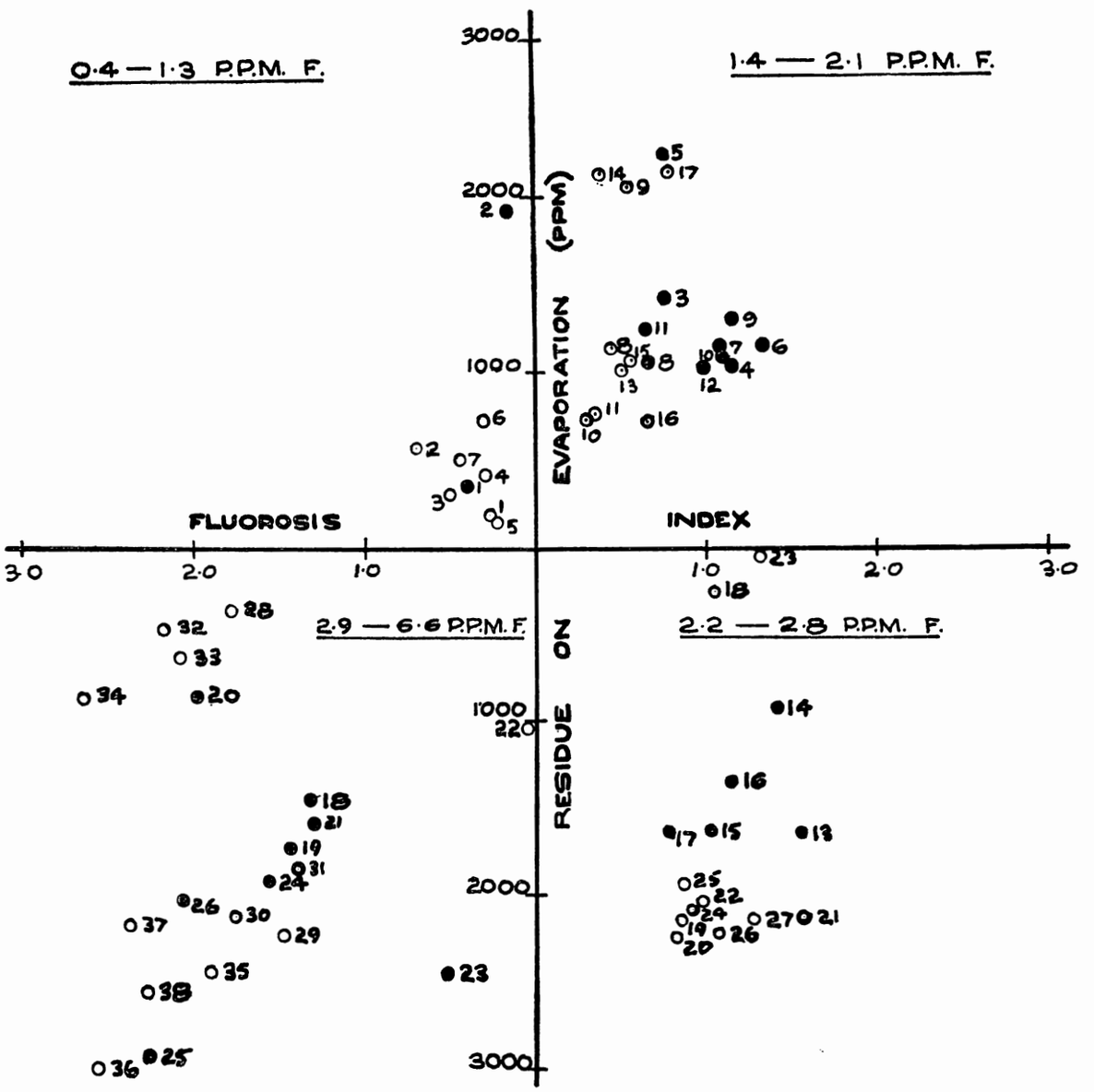
However, as so much importance was seemingly attached to this contention, I pursued it further. Professor Storey was good enough to collect from the monograph 'Fluoride Drinking Waters'[7] the recorded water data relating to fifty cities or towns in the United States and to compare them graphically with the fluorosis index for the same places as determined by Dean. Fig. X is the result. I would suggest that the results do not confirm the suggestion made and indicate no significant trend.

704. One final comment. Evanston uses lake water which contains approximately 15 ppm calcium[16] which is comparable with Hobart and Launceston. In that city the extent of dental fluorosis after fourteen years fluoridation in 920 12- to 14-year-old children examined was 774

FIG. X

EXHIBIT 843

DIAGRAM PREPARED FROM A GRAPH SUBMITTED BY PROF. E. STOREY TO SHOW LACK OF ANY SIGNIFICANT CORRELATION OR TREND BETWEEN THE FLUOROSIS INDEX AND PARTS OF HARDNESS OF COMMUNAL DRINKING WATERS IN OVER 50 AMERICAN CITIES STUDIED BY DEATHERAGE AND DEAN.



INDEX OF PLACES, FLUORIDE LEVEL, AND RESIDUE ON EVAPORATION IN PPM.

DEATHERAGE ●

1. Geneva	1.0	350
2. Kewanee	1.0	1900
3. Bradford	1.5	1420
4. Apha	1.6	1040
5. Toluca	1.6	2210
6. Toulon	1.6	1150
7. Wyoming	1.6	1140
8. Alexis	1.9	1070
9. Aledo	1.9	1300
10. Viola	1.9	1120
11. Elmhurst	2.0	1260
12. Canton	2.1	2030
13. Minonk	2.2	1680
14. Woodhull	2.2	920
15. Princeville	2.4	1630
16. Abingdon	2.5	1340
17. Farmington	2.5	1630
18. Elmwood	3.0	1490
19. Glasford	3.0	1720
20. Kempton	3.1	880
21. Odell	3.1	1670
22. Cullom	3.2	1010
23. Saunemin	3.8	2480
24. Bushnell	4.0	1920
25. Ipava	4.1	2980
26. Bureau	6.0	2030

DEAN ○

1. Elgin	0.4	180
2. Pueblo	0.6	588
3. Big Springs, Texas	0.7	314
4. Junction City, Kansas	0.7	401
5. Mullins, 5th Cav.	0.9	172
6. Aurora	1.2	730
7. Joliet	1.2	566
8. East Moline Ill.	1.4	1129
9. Howard	1.4	2176
10. Maywood	1.4	723
11. Webster City, Iowa	1.5	729
12. Aberdeen (Wash. St.)	1.6	2183
13. Monmouth	1.7	1032
14. Aberdeen (W. Hill)	1.7	2145
15. Galesburg Ill.	1.8	1080
16. Elmhurst	1.8	738
17. Iroquois	2.1	2116
18. Clovis	2.2	266
19. Miller	2.2	2152
20. Leola	2.3	2228
21. Wolsey	2.4	2132
22. Gettysburg	2.4	2062
23. Colorado Springs	2.5	46.5
24. Carthage	2.6	2143
25. Onida	2.6	1988
26. Redfield	2.7	2260
27. Ipswich	2.8	2192
28. Plainview	2.9	390
29. Doland	2.9	2276
30. St Lawrence	2.9	2134
31. Platte	3.0	1894
32. Amarollo	3.9	468
33. Conway	4.0	633
34. Lubbock	4.4	882
35. Pierpoint	4.7	2459
36. Hecla	5.0	3000
37. Lake Preston	5.9	2193
38. Britten	6.6	2568

(84.13%) normal, 76 (8.26%) questionable, 66 (7.17%) very mild and 4 (0.44%) mild. I quote from the Report[17] 'Not a single opacity was observed in the deciduous dentition. None of the subjects who had dental fluorosis had the type that could be recognised during a social conversation. The most common areas of involvement were on the cuspal ridges of the bicuspid teeth.'.

705. The suggestion that fluoridation of 'soft' waters may bring in its train an excessive degree of dental fluorosis or other undesirable side effects seems to lack both chemical and epidemiological foundation.

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CHAPTER 42. FLUORINATED PHARMACEUTICALS, ORGANIC AND INORGANIC

706. Fluorine in one form or another appears in a variety of drugs and pharmaceutical products. It has been suggested that in individual cases there could be a danger that total intake of fluoride may be boosted to undesirable levels by the prolonged use of fluorine-containing pharmaceuticals.

707. The argument has particular reference to organic forms of fluorine. Its inorganic salts, or the fluoride ion may be used dentally in caries prevention, but it should, I think, be obvious that fluoride supplements for children are unnecessary in the presence of fluoridation and should be avoided. This applies not only to straight-out fluoride tablets but to the use of a number of fluorinated vitamin and calcium supplements such as are reported by Feltman and Kosel[1] to be in use in America. They no doubt have their analogues in the Australian pharmacopoeia but identification is not always easy. As these are generally prescription items their interdiction in a fluoridated area should present no problems, but the suggestion does emerge from the authors' comments—and I commend it—that the fluoride content of such drugs should be stated on the label and be available to the physician.

708. For infants too, fluorinated dentifrices pose a slight theoretical risk, though the possibility of any significant contribution to systemic uptake so as to produce dental fluorosis seems very remote[2]. For older children and adults the risk of any significant ingestion is so small[3, 25] that their continued use may be regarded as beneficial[28]. Other forms of topical application are not contra-indicated and may by surface uptake reinforce the effect of fluoridation[25, 26, 27]. Apart from veterinary medicine the only other current therapeutic use of inorganic fluoride that has been brought to my notice is in connection with the treatment of osteoporosis and other bone disorders. This has been commented upon separately. The dosages employed are such that the supplementation obtained from dietary fluoride is relatively minor and innocuous.

709. There is, however, an increasing use in pharmacy and industry of fluorine in organic compounds. Industrial hazards are not within the scope of this Report, but have been briefly noted in a previous chapter. Where habitual ingestion or other significant exposure is suspected as a result of industrial processes in which fluorine is used or released it should be controlled by appropriate measures, as should other occasional hazards such as may exist in connection with processes involving the use of teflon (Polytetrafluorethylene)[4]. The remedy in such cases lies in the field of industrial safety regulation and should not be dependent on water fluoridation.

710. Dealing with pharmaceuticals it will be appreciated that for organic compounds of fluorine to become physiologically available as fluoride and contribute to total fluoride intake there must be a metabolic breakdown of the organic molecule leading to a release of the free ion. Even if this can occur the quantity absorbed and the period of administration must be such as to be meaningful within the range of toxic and non-toxic levels discussed in previous paragraphs.

711. I am not concerned to examine and it does not seem in any way relevant to the matter of fluoridation, the general biological effects of organic fluorine compounds. Apart from three well-known exceptions in the botanical kingdom, they do not exist in nature and they are not synthesised within the body. Some are extremely toxic, some just as inert. To the former group belongs the fluoracetates of which Compound 1080 and the phosphofluoridates of which the nerve gases are examples (both mentioned by objectors in evidence); to the latter the familiar teflon of everyday surgical and domestic use. In the case of the toxic compounds it is incontrovertibly established I think and it is accepted on all sides that the fluoride in such compounds exerts no separate effect, but that the toxic quality of the compound is that of the molecule as a whole[5, 6].

712. The question therefore is one of human metabolism. How far is it possible to assume or infer that any such compounds may be split within the body by metabolic processes so as to release the fluorine content and make it available as a separate physiological agent, and if so are the quantities likely to be significant?

713. Notwithstanding the potentially enormous number of organic compounds containing fluorine it seems that such a possibility, if it does exist, can exist only for a very limited class. Owing to the strength of the fluorine carbon bond, in two of the largest groups the fluoracetates and the fluorocarbons, the evidence is clear that chemically it is either impossible or the possibility so remote that it should be disregarded[6, 7]. In other words the fluorine present will be metabolically inert. However, the possibility of metabolic breakdown is admitted for other groups such as the phosphofluoridates. The toxicity of this group is very high and in the absence of any information as to therapeutic uses their relevance is perhaps questionable.

714. I have had produced to me a communication from Professor Burgstahler[8], which I accept, that there is evidence that *some* (emphasis is that of the authority cited) inorganic fluoride may be liberated in the metabolism of fluoroglumatic acid and fluoroproline and 'certain drugs' and the speculation has been advanced by another authority[6] that it may be possible in other groups in which the carbon fluorine bonds are weakened enough by 'suitable chemical substitution to allow the halogen to be split off within the body. Such compounds could be, for example, fluorine-containing aromatics like nitrofluorobenzenes or 1-fluoroanthraquinone, in both of which the C-F bond is loose'[6, 9].

715. The evidence does not establish that it *will* happen for any of the fluorinated drugs in current use that were mentioned to me. However, it is admitted that beyond the generally accepted fact of the strength of the carbon fluoride bond which would tend to prevent breakdown and ion release, little is known of the metabolism of specific products. Hence it may be wiser to assume that in the case of some products at least it is potentially possible that in the course of metabolism physiologically available fluoride ions could be released. But even if, against the general trend of the evidence, this possibility is assumed, it remains to assess its significance.

716. If the problem exists and to me in the light of the evidence I am about to refer to it is highly speculative, it is essentially medical. All drugs referred to in this context would and should only be available on prescription. Probably their use, certainly in the dosages mentioned to me, would be confined to adults and the dosage that would be needed to achieve even the lower limits of intake that might lead to fluorosis would be such that I can hardly conceive the possibility.

717. Waldbott[10] refers to 'certain tranquilizers and steroids, for instance, taken habitually three times a day, furnish a daily intake of from 0.8 to 1.0 mg'. In terms of total fluorine intake, even if all this is or ultimately becomes physiologically available as free ion and he admits this is not known it would not be of any great significance. Other witnesses and evidence refers to the possibility of a partial release or leaching only[11, 12] and it is difficult to see how the pharmacological action of the drug could be maintained in the presence of a complete metabolic breakdown.

718. When pursued the suggestion narrowed itself to three classes of compounds. The inorganic class of fluoride supplement, whether with or without vitamins and other mineral salts, has already been referred to. Bone meal supplements probably belong to this class but they are not an efficient source of fluoride due to the insoluble nature of their salts. The next is important containing both diuretics and tranquillizers in common use. Both belong to the trifluoromethyl group of compounds and their importance consists in the fact that proprietary brands may contain as much as 17% and 16% approximately of fluorine[6]. 'Aprinox' (13.6%)[13] was the example referred to in evidence of the first and 'Stelazine' (14%)[14] of the second. Of course total intake would be determined by dosage and the possibility of fluorotic effects, if any, would have to be related to the period of time over which taken. At a dosage of 5 mg a day which would seem to be the daily dosage recommended for the diuretic mentioned the total intake of fluorine would still only be .68 mg. I obtained no consistent figure for dosages of 'Stelazine' but even in the abnormally high dosages (e.g., 30 mg a day as against a normal dosage of 2 mg[15]) which some witnesses thought might be employed in mental hospitals, the total fluorine intake would not exceed 3 or 4 mg a day. To constitute a danger, even if totally available and it is in my opinion plain it is not, such a dosage would have to be prolonged for many years.

719. In fact, no one has claimed to demonstrate that such compounds will yield free ion. Buu-Hoi[6] cited by Professor Amies suggests only that before such drugs are put to use 'estimations be made of the degree of stability of the carbon fluorine bond during their passage through the animal body'. In fact, inquiries made on behalf of the Commission reveal this has been done[13, 14, 16, 17] and they confirm the stability of the trifluoromethyl group in the body and that specific studies have failed to detect any release of free ion as a result of ingestion of drugs of this class[18].

720. The third class of drug mentioned was the cortico steroids which may be fluorinated to increase potency. These as a potential source of objection were but faintly pressed. Professor Amies saw no dangers associated with their use attributable to fluoride[19] and I have no other evidence. There is nothing in the literature that I can find to suggest that the carbon fluorine bond may not be sufficiently stable to prevent any augmentation of body fluoride from such a source, and a great deal to the contrary[20, 21]. Steyn[22] suggests that the action of cortico steroids may induce a state of negative calcium balance and so aggravate fluorosis. This was not discussed before me except in Counsel's submission[23] but as a suggestion of ill effects caused or potentiated by fluoride it is technically reviewed by the South African Commission and dismissed[24].

721. With great respect to those who broached the matter, in the case of organic compounds of fluorine I find the whole discussion completely speculative insofar as it seeks to suggest a risk of any significant augmentation of dietary fluoride as a result of the use of such drugs. In my opinion the suggestion lacks evidentiary substance. I think it very proper to direct attention to the fact that there are some inorganic compounds used in proprietary fluoride supplements and vitamin preparations which should be controlled by restricting their use except upon prescription and by making it mandatory for the composition of the preparation to be disclosed on the label. The same precautions could possibly be taken for organic compounds, though the reasons for doing so do not possess the same cogency. These precautions taken, I see no real risk that fluorine-containing drugs and pharmaceuticals could so augment individual dietary intakes as to produce deleterious effects[24].

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CHAPTER 43. WHETHER THERE IS A BENEFIT TO ADULTS

Section (a)—Dental

722. In a previous Chapter (Ch. 9 secs. (e), (f) and (g)) the methods whereby fluoride is incorporated in teeth were discussed. Systemic uptake by accretion is not significant after the eighth year, being restricted thereafter to the third molars (for a short period) and to such subsidiary matters as cementum and secondary dentine. Topical uptake by ion exchange is substantial while the teeth in the pre-eruptive stage are exposed to the body fluids, but the question arises whether it continues and if so is it important after the teeth have erupted from the gum. This (excepting the third molars) should be complete by the attainment of the thirteenth year. These questions lie at the root of a commonly expressed objection that fluoridation serves no useful purpose beyond that age and that as a result adolescents and adults are unnecessarily exposed to something they could do without.

723. I believe the objection to be ill founded. I think it is clear that the process of ion exchange is continuous throughout life though its rate may diminish with age[1, 2]. In the immediate post eruptive stage the evidence of several expert witnesses was that the tooth enamel is relatively

permeable and subject to the process of ion exchange, and that in this period of maturation the fluoride uptake is substantial. As this stage is completed the rate of uptake diminishes, but it nevertheless continues[3, 4]. Jackson and Weidman (1959)[4] showed that at three different levels of ingestion there was a consistent relationship between average enamel fluoride content, the level of fluoride in the water and age. This as a general pattern has been confirmed[5]. Ion exchange at the enamel surface is the only mechanism whereby this result could be achieved and it is, I think, accepted. Brudevold has estimated that the presence of 1 ppm of fluoride in the water supply causes an increment of about 300 ppm in the surface enamel over that occurring in teeth from low fluoride areas[6].

724. My understanding of the evidence is that the process of ion exchange is not a one-way route; fluoride is both gained from and lost to the oral fluids as a part of the same process. Further, within the crystal structure of the enamel there is also some migration of fluoride from surface layers to deeper elements which are less well supplied, the surface elements being replenished by the oral fluid. Similarly attrition may play a part. As surface crystals and surface ions which are loosely bound are removed by brushing or wear, layers of apatite in which the fluoride content is less, are directly exposed to the oral fluids[7, 8]. On the other hand if the fluoride gradient of the oral fluid falls, then it is postulated that fluoride ions can be lost to the saliva from the surface enamel and replaced by other ions[1]. In this manner the protection gained by initial fluoridation can be slowly lost or diminished if it is discontinued.

725. I would refer to the South African Report for a more technical description of the process[2].

> 'Hydroxyapatite in enamel and dentine seems to act as an ion exchanger. The exchange of ions between the apatite and the surrounding fluids can involve the hydration shell, the crystal surface and the body of the crystal. Using ^{18}F Hardwick and Fremlin concluded that radioactive fluoride initially exchanges more rapidly with non-radioactive fluoride than with other groups, thus iso-ionic exchange occurs more readily than hetero-ionic exchange. In a period of six hours ^{18}F will penetrate sound enamel to a depth of a few microns. In contradistinction to the fluoride that enters the crystal lattice, that in the hydration shell and, possibly, the fluoride of the crystal surface, is readily available for ionic exchange. Thus a moment-to-moment interchange of ions between enamel and oral fluid may occur. If this exchange is governed by diffusion according to concentration gradients, then reduction in the fluoride concentration of the oral fluids will provide circumstances which favour the diffusion of fluoride into the oral fluids.
>
> 'In this way the fluoride content of the enamel, especially that in intimate contact with the oral fluids, may be decreased by passing into the oral fluids or continuing to diffuse to the deeper layers of the enamel, where there is less fluoride.
>
> 'In this way the main reduction will occur in the apatite hydration shell but in time the fluoride in the crystal lattice may also be substituted.
>
> 'On the other hand raising the fluoride content of the oral fluids (fluoridated water) will tend to increase the fluoride content of enamel by a similar maximum.'

726. The oral fluid for this purpose includes both the saliva and ingested fluids. The homeostatic mechanisms which regulate the fluoride content of the plasma seem also to be effective in the case of the saliva, but the evidence as to how far the saliva will reflect any increased levels of dietary uptake up to 1.8 ppm F is not clear[9, 10, 11]. However, while there is some doubt as to the quantitative role that saliva plays in the accumulation of fluoride in the surface enamel, the long term bathing effects on the enamel by the continuous saliva flow 'should not be dismissed'[3]. Both the saliva and ingested fluids have free access to erupted tooth surfaces and also to a minor extent to the dentine where it is exposed through natural fissures or carious lesions, but it would seem that as vehicles for the fluoride ion the part played by ingested fluids could be the more important.

727. An important aspect of post eruptive fluoride uptake is that apart from the uptake by intact enamel surfaces there is a relatively more intensive uptake in enamel imperfections including early cavities[12]. As Jenkins says in the paper cited, 'This suggests that under normal physiologic conditions fluoride might accumulate in early cavities and exert its effect just where it could be most beneficial'.

728. Further, not only does it act on incipient lesions by reducing enamel solubility, but it also promotes remineralisation in such sites by favouring the precipitation of calcium phosphate from the saliva[12, 13, 14, 15]. This, however, though important[14] is a retardant and not a curative effect.

729. Dental plaque has already been referred to. It has the capacity of accumulating fluoride in high concentrations up to 60 ppm and perhaps higher. Thus enriched it plays an important role in inhibiting caries probably by preventing the bacterial fermentation of carbohydrate remnants which leads to acid production, though the mechanism which is obviously complex has not been fully delineated. Plaque is constantly being removed and renewed and depends primarily for its fluoride uptake on the oral fluids though some may be gained from the enamel surface which it covers[18]. It has been shown that the plaque concentration of fluoride in children drinking water containing 2 ppm of fluoride was nearly double that of children drinking water with a lower concentration and it would seem that a relationship between the two is involved[17].

730. The mechanisms discussed constitute theoretical explanations of the means whereby the continued drinking of fluoridated water may benefit adolescents and adults. The delineation of this benefit by epidemiological means is difficult because of the inherent weakness of the DMF index in reflecting in adults the total state of dental health and not necessarily caries experience. This has been discussed previously (Ch. 12 (a) and (b) and Ch. 31).

731. The matter has been approached in the evidence and in the literature from two angles—

(*a*) Whether there is a continuing topical effect; and

(*b*) Whether the beneficial effect gained in childhood is retained in adult life or whether despite continuing fluoridation the initial effect is a delaying one only.

732. These are perhaps different aspects of the same question and the evidence overlapped, but it is convenient to consider the arguments raised under both heads.

733. As to the first question the mechanisms have already been described which suggest the probability but in some of the early studies designed to test the matter epidemiologically the results are sometimes equivocal and at best unimpressive. The South African Report[19] contains an interesting composite table in which early results for Grand Rapids (fluoridated in 1945) Aurora (naturally fluoridated at 1.2 ppm) and Muskegon (fluoridated in 1951) are compared. Taking that table and referring to the original studies[20, 21, 22], the figures would appear to show a clear trend in favour of a post eruptive topical effect in Grand Rapids. However, in the case of Muskegon, it is so slight (after four years of fluoridation) as hardly to be significant. It is further complicated by the fact that in the five prefluoride years in Muskegon there were fluctuations in the DMF rates[22] that are difficult to understand except on the basis of some facts or factors other than fluoride which is unaccounted for.

734. Other early studies[23, 24, 25, 26] have been the subject of debate. I think they are difficult to evaluate because of the crudity of the DMF index or other indices of teeth (not surfaces) which they employ and because of their failure to distinguish between those teeth as well as surfaces which are preferentially attacked by caries and others which are not[27]. They also suffer, as was pointed out in cross-examination from a general failure to distinguish between post calcification and post eruptive stages of dental life. However, the hypothesis of a post eruptive topical effect (and the authors of those early studies claimed no more) must I think be taken as deriving substantial confirmation from later and more selective studies[28, 29, 30, 31, 32], though there are two studies which show only a minimal or no benefit[33, 34].

735. However, the matter has been approached from another aspect in Russell's studies of migrant children, i.e. children who had lived in fluoridated areas during the period of calcification but had thereafter migrated to nonfluoride areas. In these he was able to show that the protection afforded by fluoridation tended to be lost unless it was continued post eruptively. Similar effects were observed in the case of a city which discontinued fluoridation after four years[35]. Evidence given as to clinical experience in relation to Italian and Greek migrants who, after leaving high fluoride areas in their native countries have arrived in Tasmania with either caries free or low DMF mouths but who have, within five years of residence in this State, reverted to the caries picture of the rest of the population would tend to the same end[36,37].

736. I think the evidence supports the conclusion that while it is the fluoride built into the tooth structure during formation and calcification which is of prime importance, there is a continuing topical uptake both pre and post eruptive which is of importance to the maintenance of dental health. It would seem that the emphasis should be on maintenance. Initial protection as a result of post eruptive surface uptake only will be low and in those teeth surfaces such as pits and fissures in first molars which are more prone to caries attack it will be minimal depending on the interval between eruption and exposure. It is also relevant to note that this topical uptake is more likely to be attained by the higher fluoride concentrations to be found in drinking water than those in saliva, though the latter might have some long term effect.

737. I turn now to the second question, whether the protection acquired in childhood is retained throughout life or whether despite continuing fluoridation it is gradually lost so that the initial benefit is at best only a delaying effect. That protection will be lost if fluoridation ceases has already been suggested. The question is whether it is retained if fluoridation continues. If it does it would serve to confirm the supposition of post eruptive benefit as well.

738. Russell and Elvove[38] made a study of the adult population of Colorado Springs which had had naturally fluoridated water for over forty years and compared it with a similar group of adults in the nearby unfluoridated city of Boulder. The age adjusted DMF rate for Colorado Springs was 7.20 and for Boulder 17.76. The House of Delegates of the American Medical Association agreed with the following conclusion:—

> 'This indicates that with fluoride there is not a mere postponement but actually a much lower caries rate, at least until the person reaches 44 years of age, and in accordance with the trend, probably much longer.'[39].

Another report in relation to the same two cities can be cited[40]:—

> 'The benefits accrued during childhood are carried over into adulthood as shown by a comparison of missing teeth of adults in Boulder, Colorado (no fluoride in the drinking water), and Colorado Springs (2.5 ppm of fluoride in the drinking water). At age 40-44 the loss of teeth is less than four permanent teeth per person in Colorado Springs, while it averages about 16 permanent teeth per person in Boulder, Colorado. The ratio of tooth loss is four to five times greater, at all times in the non-fluoride area than in the fluoride area from age 20 to 44.'.

739. Similar conclusions of lasting benefit were arrived at by Deatherage (1943)[24] and by Englander and Wallace[41]. In the latter study a comparison was made between the inhabitants of neighbouring cities Aurora (1.2 ppm fluoride) and Rockford (0.1 ppm fluoride). I quote from the report at p. 892—

> 'Subjects from Aurora had an approximate average of 10 decayed, missing, or filled teeth, and 22 decayed, missing or filled tooth surfaces, while their Rockford counterparts had 17 DMF teeth and 43 DMF surfaces. Thus, the dental caries experience of Aurorans was roughly 40 to 50 percent less when evaluated by teeth and surfaces. These differences might have been greater if a random sample had been examined. Persons with fewer than 10 teeth were excluded from examination, and as a result, many more persons were eliminated in Rockford than in Aurora. About seven times as many edentulous persons were noted in Rockford as in Aurora.'

and see also Backer Dirks (1962)[28].

740. Similar results have been claimed in Russia[42], in Hungary[38], in Czechoslovakia[43] and in Switzerland[44].

741. The suggestion that the protection obtained in childhood is not maintained in adult life and that it commences to be lost either at eruption or soon thereafter so that the subsequent caries increment approximates or even exceeds that of the non-exposed population finds its support (exclusively according to Brudevold)[45] in a study made in relation to North (unfluoridated) and South Shields (1.4 ppm) in England by Weaver[46, 47, 48]. As a result of his comparison between DMF rates (teeth) of children between the ages of 12 to 17 Weaver (1944)[47] (1948)[48] suggested, though there was approximately a 50% difference in DMF rates between the same age groups in the two towns, that the inhibiting effect of fluoride was only to delay the onset of caries by approximately three years, e.g. the fifteen year olds in the fluoridated area had the same DMF rate as the twelve year olds from the control area. He went on to make an examination of mothers in both areas and arrived at the conclusion that the period of delay for this class of adults was probably no more than five years.

742. The samples were small—in fact too small—and the result is complicated because he was endeavouring to compare the overall dental state and not the caries experience of his two groups. Hence, as he says, while teeth may have been lost due to periodontal disease or because of a preference for a full denture or dentures to partial, it was immaterial to his study whether the teeth lost were due to caries or not. He himself said in 1944—[47]

> 'It would be unwise to make generalisations based on these figures and to argue that the caries inhibiting factors can do no more than postpone caries for about 3 years in childhood and for about 5 years in adult life.'

However, this has been done by others for him and certainly when he referred to the matter again in 1948 he was not so cautious[48].

743. He made a further investigation of the same two areas in 1949[49]. This was primarily to study whether wartime diet had had any effect on the prevalence of caries. This was confirmed and this factor alone makes dangerous any comparison between caries rates found in 1943 and 1949. This time he extended his study to West Hartlepool (2 ppm fluoride) which had not been included in the 1943 study. For twelve year old children the results were as follows:—

	North Shields (No Fluoride)		*South Shields 1.4 ppm Fluoride*		*West Hartlepool 2 ppm Fluoride*	
	1943	1949	1943	1949	1943	1949
Average DMF per child	4.3	2.4	2.4	1.3	....	0.96
Percent with no DMF permanent teeth	4.8	26.9	25.8	50.6	....	59.8

744. In 1949 he also made an examination of 100 mothers in the West Hartlepool area. Unfortunately similar examinations for North and South Shields were not repeated. The combined results of the two studies can be tabulated as follows (Table XXXI) :—

TABLE XXXI

DMF TEETH IN MOTHERS IN NORTH AND SOUTH SHIELDS AND WEST HARTLEPOOL (1943 STUDY)

Age Group (years)	*Under* 20	20-24	25-29	30-34	35-39	40 and over
North Shields (no fluoridation)						
Number of mothers	1	25	33	22	12	7
Average number of DMF teeth per mother	6	11	15	17	18	19
South Shields (1.4 ppm fluoride)						
Number of mothers	4	32	19	20	16	9
Average number of DMF teeth per mother	5	7	9	15	17	17
West Hartlepool (2 ppm fluoride) (1949 study)						
Number of mothers	2	21	39	19	14	5
Average number of DMF teeth per mother	3.5	7.5	7.9	8.4	11.9	17.2

Taken from Weaver[47, 49].

745. No comparison between the West Hartlepool and North and South Shields results is possible because of the time difference between the examinations. But Weaver compares the West Hartlepool mothers with the children from the same area and concludes[49]:

> 'The caries incidence figures in Table VI show that the protection afforded by fluorine is not lasting. Of the 100 mothers seen, 37 had 10 or more DMF teeth, though it may be taken as fairly certain that not all the missing teeth had been carious. Some may have been extracted for aesthetic reasons, some for parodontal disease, and some for denture planning reasons. The fact remains, however, that there was a great difference between the children and the adults—for example, the percentage of caries-free mouths in the children of 12 years was approximately 60; in the mothers it was 5.

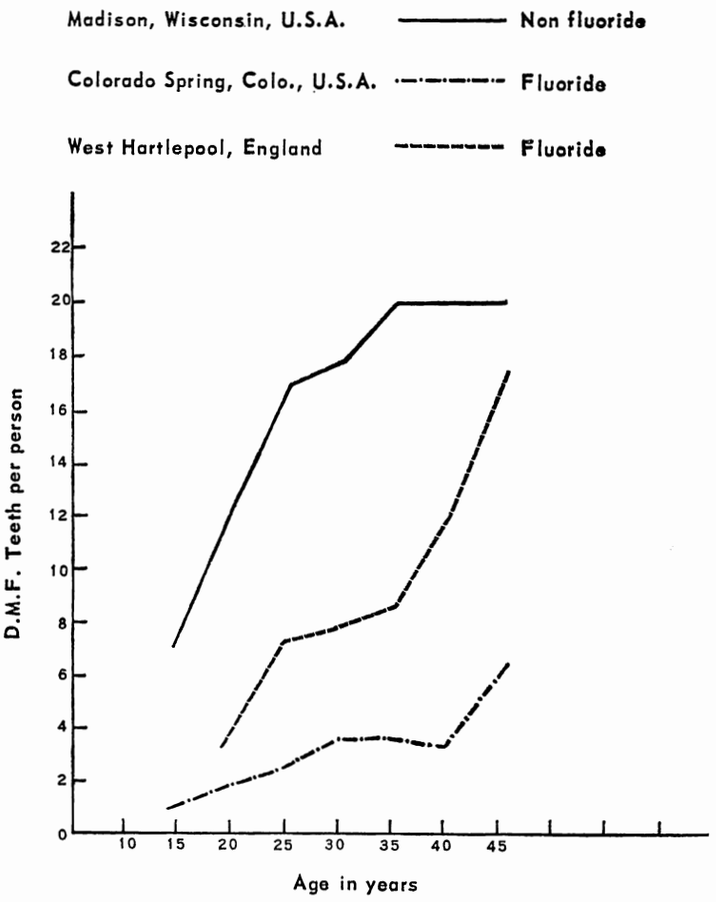
No other class of adults was studied. The total sample in each case was 100 and as there were six age groups the numbers studied in each group were small. By comparison with young Australian males for instance, in the 20-21 year old age group (see Ch. 13, *supra*) all the DMF figures are extraordinarily low, though the comparison would not be valid for several reasons. It is not possible to refine the DMF figures given so as to give a more accurate picture of caries severity apart from prevalence. I suggest strongly that the data is too meagre to support the generalisation that the protection afforded by fluoride is not a lasting one[45].

746. No subsequent investigation has confirmed Weaver at least to the extent of the number of years difference that he suggests. Martin[50] compared Weaver's West Hartlepool figures for mothers with the American data for Colorado Springs and Madison (adults)[51] in Fig. XI. This shows a marked difference between the English and American results. Because the classes are not essentially the same the comparison is a crude one but the upward swing that occurs between the thirty-fifth and fortieth birthdays is marked in both cases, but how far this is due to caries or to periodontal disease or other causes or to both is not really determined.

747. It is to be remembered that the yearly incremental rate of caries attack is ultimately proportional to the number of surfaces at risk. As a matter of yearly incidence it may well increase

with age in the case of those subjects who have attained adult life with relatively caries free mouths and therefore with more surfaces at risk as against those who have at a younger age already had most surfaces attacked or even lost their teeth. Weaver's figures, at best a crude DMF count of teeth and not of surfaces, do not reflect whether one or more surfaces are attacked or the severity of the lesions experienced. This is important in comparing adult groups[52, 53, 6].

FIG. XI



Relation between D.M.F. teeth and age of persons living in specified areas

748. Later more extensive studies by Forrest *et al.* (1951) in England gave the difference in 'postponement' as ten years as against Weaver's five as Table XXXII shows. Again these results differ from comparable U.S.A. studies.

749. The United Kingdom Mission of 1952 (from whose report this table is taken) made a special study of these differences. They drew attention to the fact that in the English groups the percentage of missing teeth (which would be the index influenced by periodontal disease) among the total DMF teeth rose with age from 41% to 95% in the low fluoride areas to 36% to 88% in the high fluoride areas. By contrast the same percentages in Boulder rose from 9% to 56% and in Colorado Springs from 11% to 30%. In American investigations a distinction was made between teeth extracted for caries and those extracted for other reasons.

> 'This differentiation was not possible in England but the steeply rising extraction (M) rate suggests that many teeth were removed for reasons other than caries, such as paradontal disease or for denture-planning purposes. This is further supported by the fact that both D and F rates actually fell as age increased in both English groups.'[55].

750. The evidence I have taken supports the validity of this reasoning. There are no comparable studies on Australian adults, but the DMF rate for over 6,000 N.S.W. school children ascertained by Barnard *et al.*[56] was 13.91 at the age of fifteen. This is comparable to the status

of a 40-year-old West Hartlepool mother in Weaver's studies. Tasmanian children would not be lower and may even be higher. The DMF figure for fifty-two Tasmanian National Servicemen discharged after two years' service in the Army where free dental treatment was available, after excluding twelve (or 26%) because they were fully edentulous, was 21.7[57].

TABLE XXXII

INCIDENCE OF DENTAL CARIES AMONG ADULTS IN ENGLAND AND THE UNITED STATES OF AMERICA, SHOWING THE AVERAGE NUMBER OF DECAYED, MISSING AND FILLED TEETH PER SUBJECT

England:

Age in Years	*Combined Low Fluoride Areas* (0.07-0.3 *ppm F*)					*Combined High Fluoride Areas* (0.82-1.45 *ppm F*)					*Percentage reduction in High compared with Low Fluoride Areas*
	No. Exam.	*D.*	*M.*	*F.*	*DMF Rate*	*No. Exam.*	*D.*	*M.*	*F.*	*DMF Rate*	
21-25	92	7.7	6.7	1.7	16.1	91	4.6	3.6	1.8	10.0	38.0
26-30	107	5.8	11.9	1.6	19.3	69	3.6	6.7	2.2	12.5	35.0
31-35	40	5.2	14.6	1.6	21.4	61	3.7	11.1	1.4	16.2	25.0
36-40	30	3.4	18.4	1.0	22.8	23	2.9	15.3	1.0	19.2	16.0
41-45	5	1.4	25.0	0.0	26.4	7	1.8	19.4	0.8	22.0	17.0
			All ages		19.1			All ages		13.4	

U.S.A.*

	Boulder (0.025 *ppm F.*)					*Colorado Springs* (2.5 *ppm F.*)					
20-24	51	1.1	1.2	11.7	14.0	72	0.2	0.6	4.6	5.4	61.5
25-29	41	1.2	2.5	12.8	16.5	101	0.3	0.9	5.3	6.5	60.6
30-34	29	1.1	4.0	13.1	18.3	82	0.1	1.1	5.8	7.1	61.2
35-39	22	0.6	11.4	9.8	21.8	75	0.2	3.2	5.8	9.2	57.8
40-44	12	0.91	12.08	8.66	21.7	55	0.1	3.1	7.1	10.3	52.5
			All ages		17.2			All ages		7.5	

*Rates do not include the third permanent molars.

Taken from Report of the United Kingdom Mission of 1952 to North America[55].

751. Nobody has ever suggested that fluoride prevents caries in the sense of making it impossible. It has no effect on periodontal disease, which plays an increasingly important part in tooth mortality as age increases. It does not remove the need for dietary care or proper oral hygiene, but once those facts are appreciated, I think the conservatively expressed conclusion of the 1951 United Kingdom Mission which was 'it is not justifiable to conclude . . . that the effect of fluorides on caries is merely one of postponement' is as valid now as it was then[55].

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Section (b)—Osteoporosis

752. Apart however, from any question of dental benefit, the evidence is strong that fluoridation may benefit the adult members of the population in yet another way. Osteoporosis is a widespread and disabling disease of middle and old age. It is manifested by a loss of bony substance due generally to increased bone resorption[1]. This leads to brittleness and softness of the bones affected. Though not confined to females, in America it is estimated that one quarter of all women over the age of 50 have the disease to a clinically important degree[2]. Australian figures are not available but there is no reason to believe they are not comparable[3]. Its end results are often seen in those distressing fractures of the femurs and hips of the elderly. It is, incidentally, extremely common in countries where dietary calcium intakes are relatively high so that the suggestion follows that some other factor than calcium intake is implicated[2, 3, 1].

753. The suggestion is that fluoride plays an important part not only by promoting satisfactory calcification in bones but by maintaining it. Evidence as to the role of fluoride as a chemostat in regulating calcium deposition[4, 5], in reducing bone solubility[6] and in promoting a positive calcium balance has already been referred to (see generally Ch. 40, *supra*). In 1955 Leone and his associates[7] as a result of the 10-year Bartlett-Cameron study suggested the possibility that a high fluoride intake (in this case 8 ppm) might be beneficial in counteracting osteoporosis. In 1960 he and others[8] made a comparison of radiographic findings in 546 persons between the ages of 30 and 70 years who resided in Framingham, Mass., where the concentration of fluoride was 0.04 ppm. with those obtained in an investigation made in Bartlett and Cameron (8.00 ppm and 0.4 ppm F^- respectively). He found an unusual incidence of severe osteoporosis in the Framingham group and a considerable number of the cases detected occurred in the younger age groups, particularly in males. He also found in the Framingham group an unusually high incidence of ectopic calcification manifested in calcified abdominal nodes and calcified ligaments. On the other hand, cases of increased bone density and coarsened trabeculation were significantly fewer than those found in Bartlett, but were comparable to those found in Cameron.

754. A 1966 study by Bernstein *et al.*[9] has compared the prevalence of osteoporosis in high (4 to 5.88 ppm) and low (0.15 to 0.3 ppm) fluoride areas in North and South Dakota. The findings were expressed in the following terms:—

> ' We would infer from these studies that fluoride in concentrations from 4 to 5.8 ppm in water (a level which produces mottling in some children) not only greatly reduces dental caries but materially and significantly lessens the prevalence of osteoporosis and

collapsed vertebrae, more in women than in men. There is overwhelming evidence that fluoride induces crystal growth and perfection in the enamel of teeth, and, likewise, the same process occurs in the hydroxyapatite of bone. The enlarged and more perfect bone crystal may be less susceptible to resorption, accounting for our observations.'.

755. However, while the Framingham and North Dakota studies referred to indicate that fluoridation from 4 ppm to 8 ppm may be expected significantly to lessen the incidence of osteoporosis Geever *et al.*[10] found no evidence of increased bone density or osteosclerosis where the water supply was less than 4 ppm fluoride and this was substantially confirmed by Zipkin *et al.*[11] though the latter found that the concentration of fluoride in the bones increased in a linear fashion with an increase in the drinking water up to 4 ppm even in quite elderly subjects whose initial exposure was late in life. These findings do not necessarily contradict a hypothesis of benefit in relation to osteoporosis as an increase in bone fluoride content alone without any apparent increase in density could be beneficial in enhancing calcification and lessening solubility[11, 12].

756. However, the need is for studies specifically directed to considering the relation between the incidence of osteoporosis and fluoride at 1 ppm; but I believe that no such studies have as yet been done. Until this has been done the evidence while highly suggestive of benefit cannot be taken to establish it with certainty. It may even be that from this aspect the optimum level for adults is higher than that for children[2, 13] for whom it is determined by the threat of dental fluorosis which is not relevant to adults.

757. One final point. In the human body the parathyroid glands are concerned with the mechanism of bone resorption and the maintenance of serum calcium balance. Though little reference was made to them during the evidence, as, in this connection they constitute an obvious query, I think I should record that both the New Zealand[14] and the South African Commissions[15] considered studies relative to any possible adverse effects that fluoride may have upon their physiological role with negative results.

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Section (c)—Calcification of the Aorta

758. In their 1966 study of groups from high and low fluoride areas in North and South Dakota, Bernstein and his associates[1] recorded a further unexpected finding:—

> ' An incidental finding, statistically highly significant in men and potentially of considerable importance, was the reduction in calcification of the aorta in both sexes in the high-fluoride area '.

759. This last finding is consistent with the findings as to ectopic calcification in the Framingham[2] study, with evidence given to the Commission by Professors Storey and Holdsworth in particular and with the prior suggestion of Bernstein himself[3] to the effect that not only does fluoride promote and maintain proper calcification of bone, but by inhibiting pathological resorption it lessens calcium loss and ectopic deposition in sites particularly susceptible such as the aorta. However, while suggestive of an important benefit, this should, I think, be regarded as a preliminary observation awaiting confirmation.

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Section (d)—Conclusion

760. In summary I think that dental benefits of early fluoridation persist into adult life. On the evidence I think there is a continuing topical uptake of fluoride which is dentally beneficial and that some of the benefits of early fluoridation will be lost or diminished if it is discontinued. I discount the suggestion that the benefits accruing in childhood cease with adolescence and that fluoridation confers no more than a temporary delay of small duration in the total caries incidence. There is a probability that other physiological benefits may accrue from fluoridation, but this has not yet been confirmed by studies specifically related to fluoridation at 1 ppm. However, the relative importance of these matters must not be overstressed. The case made for fluoridation is the dental benefit to children, the possibility or even the probability of benefit to adults is a concurrent but not in the present state of our knowledge a determining factor.

PART J—MISCELLANEOUS OBJECTIONS

CHAPTER 44. SOCIO-POLITICAL OBJECTIONS

Section (a)—Introductory

761. This is a clumsy title. I believe it to be more accurate however than the alternative frequently adopted of 'Moral Objection'. Under this head I propose to consider briefly a number of objections which are linked by the common thread of individual liberty.

762. A preliminary point must be made. Objections of this class cannot wholly be resolved as a matter of evidence. Ultimately decisions of policy are required which belong to the body politic and not to the judiciary or any commission of inquiry. The question how far the rights of individuals in a given society should be curtailed in the public interest is one which constantly recurs in the political sphere. Basically the decision belongs nowhere else, but it is necessary to a decision that the right invoked should be identified and its extent determined. Its force as a political consideration cannot be determined without consideration of countervailing benefits, their certainty and their safety. These are matters on which I endeavour to assist without presuming to decide ultimate questions which belong elsewhere.

Section (b)—That Fluoridation is an Unwarranted Interference with the Freedom of the Individual

763. At times the arguments advanced under this head were overlaid with American echoes of 'fundamental rights' reflecting the fact that in that country as well as in others, e.g., the Republic of Eire (see for a discussion of such matters the judgments in *Ryan v. Attorney-General*[1, 2]) some principles of civilian liberty are given constitutional and statutory expression. Because of this, fluoridation proposals have often given rise to litigation in which the issue for determination has been one of construction; whether fluoridation was within the legislative power of the body proposing it. There are no 'entrenched provisions' or 'guaranteed rights' in the Constitution of this State as these phrases are understood in other parts of the world and I have not thought it necessary to refer to the cases where these topics have been pursued with great zeal but little success though I have perused a number. A number are reviewed in the New Zealand Report[3], Chapter 58, to which reference can be made for information if desired. Nor since the decision of the Privy Council in *Attorney-General of New Zealand v. Lower Hutt City Corporation* (1964), *A.C.* 1469, would it seem to me possible to challenge as *ultra vires* the action of any municipality in fluoridating communal water supplies on the grounds that it was other than pure.

764. To say that our Constitution does not so enshrine such rights is merely to point to a difference in the machinery of preservation; it is not to diminish their importance. But fundamental rights are not absolute rights; individual liberty as an abstraction should never be allowed to destroy or blur the necessary limitations which must be imposed in the interests of a viable society. May I without pedantry quote—

> 'The major problem of human society is to combine that degree of liberty without which law is tyranny with that degree of law without which liberty becomes licence' (Heracleitus).

The conflict between the individual's right to live his life as he pleases and the demands of a civilized orderly and healthy society is more apparent than real, because society in its own interests as a group has an interest in the preservation of a high degree of individual liberty. But it does presuppose a balance. The problem is not one of black or white but to choose the correct shade of grey.

765. Some opponents of fluoridation do not see it in such terms. To them the fluoridation of communal water supplies is an intolerable invasion of liberty and the term 'moral' objection is coined to express their sense of outrage. In such a context the argument tends to become emotional rather than ethical.

766. I agree with them to this extent. Fluoridation of communal water supplies has inescapable consequences for all members of the community concerned. I regard as unrealistic the suggestion that the installation of tanks or the presence of defluoridating equipment is a practical answer in the case of the individual who wishes rightly or wrongly to drink water to which supplemental fluoride has not been added. Even if the widespread provision and use of such equipment is feasible, which I doubt, there is no absolute escape for the individual who must purchase food processed with communal water and eat and drink in hotels, restaurants and the like. I think this should be recognised frankly and without evasion.

767. If adults only were concerned, the arguments raised under this head would possess much force. But fluoridation is designed to benefit children. While incidental benefits to adults are alleged as well, they are not weighty factors in the argument, and the proponents of fluoridation would not, as I understand them, seek to base their case otherwise than on the benefits which may accrue to children. If those benefits are substantial, and if, in the absence of those benefits the detriment to children is great, and I believe the evidence previously discussed establishes both propositions, then it becomes a matter of a reasonable balance between those factors and the disadvantages, including their comfort and safety, suffered by those who object.

768. In my opinion the evidence does not establish or raise any presumption of detriment to their health or comfort other than the injury to their personal sense of liberty. This may be a serious matter perhaps to those who, having been persuaded wrongly that water fluoridated at 1 ppm will injure their health, will be distressed by the difficulty of avoiding it. This could in a very small percentage have psychosomatic or neurotic effects.

769. The fact that fluoridation is intended to benefit children introduces a difficulty for those who object on the score of personal freedom. There were some who claimed that the principle they sought to invoke extended to the right of parents to determine what was or was not good for their children. Others more cautious and perhaps more knowledgeable turned the question to other matters as to whether it was effective or whether it was safe. This of course tended to confuse an argument of principle with arguments which have been dealt with elsewhere. But the fact is that in the interests of children as a class, society has long recognised and accepts without reservation as right and proper a considerable limitation on the right of parents to do what they like in regard to their children's health, education and welfare and this is so whether they are acting conscientiously or otherwise. The Tasmanian Criminal Code, Secs. 144-146 and 152, expresses the principle and there are supplemental provisions in other Statutes.

770. The fact that children of tender years are the primary object of benefit is, I would suggest, a consideration of great, in fact of preponderating weight. Such children as a class are incapable of group initiative in matters relating to their health and welfare. The evidence as to the prevalence and incidence of juvenile caries in this State makes it clear that reliance on parental responsibility is not an answer to the problem that it raises. It also suggests strongly that it is those children in particular who are least likely to have a high degree of parental care and competence directed to their health and welfare who would be most in need of the benefits that fluoridation can afford.

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Section (c)—That Fluoridation is an Interference with Religious Freedom

771. A related argument suggested that fluoridation imperilled freedom of conscience for those members of religious groups whose teachings forbade the use of drugs in illness. The suggestion was not raised directly by any religious body. However, to clarify the matter the Commission invited an official representative of the Christian Science denomination to attend. He did so and stated the views of his Church with dignity and moderation.

772. While not opposing the use of fluoride by individuals when prescribed, they were as a body opposed to the principle of communal fluoridation as representing an infringment of personal liberty. On the other hand, if fluoridation was introduced by lawful democratic processes, they would respect the law, and no Church sanction was extended to any member of the denomination who chose to use it. Whether he did so or not was regarded as a matter of individual choice and conscience. In other words, while they retained their right to disagree and to continue to disagree with the policy, they would do so within the law. ' Christian Scientists are not asking the community to reject compulsory fluoridation merely to accommodate their particular religious views '[1].

773. I thought this an essentially reasonable attitude entirely in conformity with democratic principles. It to me is plain, that fluoridation does not infringe religious freedom; it does not restrict beliefs which may be exercised as before without religious or civil sanction. The matter is more fully discussed in the Ontario Report to which reference may be made for a further discussion of principles involved if desired[2].

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Section (d)—That Fluoridation is 'Mass Medication'

774. A further objection is that fluoridation is 'mass medication', and the statement of Sir Arthur Amies is much quoted:—

> 'Fluoridation of domestic water supplies involves the administration with therapeutic intent of a chemical preparation to young and old, dentate or edentulous, well and ill, without individual examination and regardless of individual desire.'[1].

If the word 'therapeutic' were changed to 'prophylactic' I would agree that this is as accurate a description of the process as one could get and though 'medication' is doubtfully used in relation to fluoridation at 1 ppm, I think 'mass medication' or better still 'mass prophylaxis' is not an unreasonable summation of the process. Even if there be some terminological inexactness involved I think it preferable to accept the impeachment than to seek to rationalise or avoid it by verbal wrangling. Herein I would seem to differ from others who, like myself, have had the duty of inquiring into and reporting on this matter[2, 3, 4].

775. I do not propose to indulge in any semantic debate as to what is 'medication'; as to whether fluoride is a drug, a nutrient or a medicament. The last does not appear to be a term of art in the sense of having a precise and certain meaning in a technical context but even if it did I do not see that the identification of a label to be attached to the process under examination in any way advances or detracts from the merits of the matter. These should be determined not by the emotional content of any verbal category to which the subject can be ascribed, but by a dispassionate comparison of benefits and disadvantages.

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4 Republic of South Africa.—'Fluoridation'. Report of Commission of Inquiry, § 409. Govt Printer, Pretoria. 1966.

Section (e)—That Fluoridation is a Breach of 'Medical Ethics'

776. Yet another argument was directed to medical ethics, by which I understand it was meant to refer to those rules of practice and conduct which, as a matter of professional discipline, the medical profession enforces in the relations of its members to their patients and to other members of the profession. I thought the best evidence of the content of such rules would be from the representatives of the organised profession itself and I sought it. As a result I am satisfied that fluoridation as a public health measure in which no relation of physician and patient is involved is not regarded by the organised profession as involving any breach of ethics or rule of professional propriety.

Section (f)—That Fluoridation Would Constitute a 'Precedent'

777. Lastly it was said that fluoridation would constitute a precedent which could be appealed to in the future in order to permit the addition to the communal diet of other and possibly deleterious substances. Again I think it better frankly to meet the substance of the impeachment than speciously to deny it. I am not impressed by attempts to rationalise fluoridation by reference to other claimed analogies such as vaccination, chlorination, inoculation, iodised salt and other enriched foodstuffs, compulsory X-rays, blood transfusions and the like. None are exact parallels either in their own essential characteristics or in their attendant circumstances, and the argument by analogy gains no additional weight when it is applied in favour of fluoridation instead of against it. The matter should be considered in its own right and on its own merits without colourable attempts to strengthen the case from dissimilar measures introduced in the interests of public health in the past.

778. But if on a dispassionate consideration of the merits the action taken be good, does the fact that it will constitute a precedent make it bad? I suggest that it is a confusion of thought to allow values to be determined by secondary characteristics. The question is what is the benefit and what is the detriment? If it be beneficial then to reject it because it might constitute a 'precedent' is the argument of timidity.

779. There can be no question under our law and our Constitution of a binding precedent, i.e., one which compels to a future decision irrespective of the merits of the proposal then being considered. To suggest otherwise is to confuse precedents in the legal sense with precedents as mere guides to social action. In a later chapter (Ch. 51, *post*) I suggest, respectfully but emphatically, that the broad decision of policy which is required in this matter of fluoridation is one for the elected representatives of the community in Parliament assembled, and no such precedent can bind Parliament. This is fundamental to our legislative process. If beneficial measures are now to be rejected because future Parliaments may be overborne by their weight as a precedent, then with all humility I suggest there must be something wrong with our democratic processes.

780. I report the argument as one advanced for consideration. In the sense of providing a reference point in future discussions, fluoridation would constitute a precedent. In no way could it be said to be a precedent in the binding sense in which the term is used in the Law. Apart from this I can attribute no substantial weight to the argument.

781. I would conclude with a reference to Part X of the Ontario Report[1], where the matters which I have so briefly mentioned are discussed with thoroughness and clarity. I find myself generally in respectful agreement with their conclusions and recommend the study of this chapter of the Report to all who may wish to reflect further upon the matter of civil rights and liberties.

REFERENCE

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CHAPTER 45: SWEDEN—THE NORRKÖPING TRIAL: DR THEORELL

782. The fluoridation trial instituted at Norrköping in Sweden and the attitude to fluoride of Dr Hugo Theorell, a Nobel Prize winner for his research in the field of enzyme chemistry, are subjects which have been frequently raised by objectors. They serve to illustrate the ageless quality of some objections in that they tend to become static in time and to be taken up and repeated regardless of changes or modifications that the passage of time should have indicated.

783. Because, by the repetition of incomplete information, the public may be, and I think have been in this instance, misled. I propose to summarise the facts as I have been able to discover them and let them speak for themselves.

784. Sweden has some communities with water naturally fluoridated up to 7-10 ppm. In 1952 the city of Norrköping (population 90,000) commenced artificial fluoridation at 1 ppm[1]. Seven years later an investigation of DMF rates of the 7-year-old (fluoridated since birth) and of the 14-year-old (fluoridated since 7 years of age) age groups of children showed a reduction of 52.4% in the DMF surfaces for the former and 31.4% for the latter[2]. Fluoridation ceased in 1961 in the following circumstances.

785. An organisation known as the 'Health Furtherers' petitioned to have fluoridation stopped[2]. A Commission was appointed in 1952 by the Royal Medical Board 'to obtain a comprehensive view of the question with special regard to the conditions in Sweden'[3]. It reported in 1955[3]:—

> '. . . that the addition of fluorides to the drinking water would be a measure of great potential value in the prevention of the widespread disease of dental caries. In view of the fact that the scientific advisers expressed some doubt as to the harmlessness of fluoride even at the low concentration at which it was supposed to occur in drinking water, the Board considered it suitable to await the outcome of further research before fluoridation was to be more generally used in combating dental caries. The Board therefore recommended that fluoridation of domestic water-supplies be prohibited but that the Board should be empowered to grant exemption from this rule in special cases.'.

786. In 1957 the Royal Medical Board of Sweden appointed another Commission of three Professors to investigate caries prevention problems, including the action of fluorides. The Commission reviewed the World literature and by a two to one majority reported in favour of fluoridation. The reasons both of the majority and of the dissenting member are too long to summarise but follow familiar lines and are fully abstracted in the paper referred to[3]. The report of the Commission was discussed by the Scientific Council of the Royal Medical Board. The views of

the six members of this Council are also abstracted in the document. They range widely, covering not only fluoridation of water supplies and its dental and medical aspects but alternative means of administering fluoride in addition. They also discuss socio-political and economic factors as well. Not all expressed a definite recommendation, but of those that did, one was unconditionally in favour, stating that he considered the absence of any attendant risk had been verified as far as can reasonably be demanded; two were in favour on condition that systematic examinations of results were carried out and research pursued; one made no recommendation, the opinion expressed being that he was satisfied of the cariostatic effect but that in relation to the question of harmful consequences he thought 'the answer to be in the negative but here further research work has to be done'; one (Theorell) was against it for reasons connected with enzyme chemistry 'until more research work may have established that it is harmless' and one was against on the ground it might have deleterious effects 'even though experience to date does not indicate that they would be significant or that they would occur with great frequency'. He considered the fluoridation of table salt to be preferable.

787. As Professor Theorell is a focal point of this particular debate I reproduce that paragraph of the summary of his statement that is most commonly quoted—

> 'For water fluoridation at 1 ppm the short distance to toxic dosage seems to imply a serious hazard. We have even to pay attention to the great individual variation in sensitivity and in consumption of drinking water. Because of that fluoridation of tap water should not be allowed until more research work may have established that it is harmless.
>
> 'There are alternatives to water fluoridation which are as good or better. These are the topical application and the administration of fluoride by other vehicles than water.'.

788. The Royal Medical Board itself, after reviewing the opinions so far summarised and other matters, concluded[3]:—

> '. . . the inquiry carried out has shown that fluoridation of public water supplies does not involve any demonstrable health hazards even on prolonged consumption of the water. Being convinced that fluoridation of drinking water is an effective means of preventing caries in children and young persons, the Board maintains that communities which desire to start fluoridation of their domestic water supplies should be authorised to do so under the necessary technical control.',

and recommended to the Crown accordingly.

789. It is obvious the Board did not accept Professor Theorell's opinion. A statement signed on behalf of the National Board of Health and furnished by the Swedish Embassy may be taken to indicate reasons:—

> 'Professor Theorell, as an advisor of the National Board of Health, in 1958 took a sceptical attitude to the proposal of water fluoridation in Sweden. Professor Theorell based his argument mainly on theoretical considerations which in their turn were based on earlier test-tube experiments with purified enzymes and fluorides. Professor Theorell seemed to have overlooked the facts that quite different conditions prevail in the body fluids and that a concentration of 1 ppm F in the drinking water will have negligible effect on the fluoride concentration of the body fluids except the urine.'.

790. The anti-fluoridation movement then took the matter to the Supreme Administrative Court. The grounds on which the Court's intervention was sought are not clearly stated, but they apparently challenged the legal power of the city to fluoridate in much the same way and on the same grounds as have been finally disposed of for this State by the Privy Council in the *Attorney-General of New Zealand* v. *Lower Hutt City Corporation* (1964) *A.C.* 1469. They were successful. The Supreme Court ordered fluoridation to be discontinued and this was done in February 1962[4]. No appeal lay from this decision but the City petitioned the Crown for special permission to continue. In the same year the Ministry introduced a Bill into the Swedish Parliament to allow the Crown or a Crown agency to permit fluoridation in a community which requested it. This was passed on 21 November 1962, after the Minister had declared that he intended to restrict Governmental permission to the City of Norrköping for the next five years. In the following year Norrköping submitted an application in accordance with the Bill to be allowed to continue and this application was granted on 16 May 1966. Another City has submitted a similar application and I believe two others are planning similar action[1].

791. There my information stops except in relation to Professor Theorell. In 1962 he was interviewed on the subject. The report I am about to quote is taken from the British Dental Journal of 1962, Vol. 113, p. 101 and is a translation from a Swedish Journal[5]. It has been cited by the Prime Minister of the Commonwealth in Parliamentary Debate[6], is among the materials furnished by the Swedish Embassy, and I accept its accuracy.

> 'I have just drawn up another report, with Professor Jan Waldenstrom and Professor U. S. von Euler, recommending that the Norrköping experiments be continued.'
>
> 'Q. *Does this mean, Professor Theorell, that since* 1958 *you have altered your opinion as to the possible injurious effects of fluoridation?*
>
> 'A. No, if you read my 1958 report you will see that at that time I simply did not consider the time to be ripe for any general permission for the fluoridation of water supplies, and advised experiments along other lines. But now, since the Norrköping experiments have shown such good results as a 50% reduction in caries, I consider that it would be wrong to stop the experiments. Quite obviously they must go on.
>
> I stated in my 1958 report that there was a risk involved in increasing the fluorine content of certain organs of the body, and that as fluorine is an enzyme poison, the prolonged addition of fluorine to drinking water might have medical consequences. The Norrköping experiments, however, have not supported this theory. That is something we did not know four years ago. We know now that it is not dangerous.
>
> Moreover, since it has not been made public in Norrköping in which part of the town the extra fluorine has been added to the water, there is no possibility of individuals letting their imagination run away with them. That was another point on which I expressed fears in my report.
>
> Altogether I would say that time has worked in favour of the continuation of the Norrköping experiments. I now await their final results with great interest.'.

Dr Theorell has since issued the following standard reply to inquiries[7]:—

> 'The number of letters with questions on the fluoridation problem arriving here every day is too large to allow me to answer them individually. I must therefore confine myself to the following standardised statement.
>
> 'As fluorine is an enzyme poison and as in 1958 too little was known of human reactions to it I found it safer at that time to advise against a general fluoridation of tap water. Since then we have gained more practical knowledge from the experiments in Norrköping and as these have given no negative results, but on the other hand the caries frequency has gone down by 50% among the school children getting fluoridated water, I think it well worth while to continue the Norrköping experiments for another period of time. This does not include a recommendation of general fluoridation of tap water.'.

792. In relation to the last paragraph it will be recalled that Dr Theorell's statement to the Royal Medical Board in 1958 referred to alternatives to water fluoridation as 'being as good or better'[3].

793. As Sweden has been specially referred to I may be permitted a postscript on another matter. Dr Hale introduced the Commission to the works of Dr Aslander who has decided views on tooth nutrition. To him caries is a deficiency disease and his universal tooth nutrient is a bone meal supplement (made from ground up bones of all kinds with hooves and horns thrown in for good measure) to which iron, iodine and cobalt has been added up to 1.5 to 2 gm a day and the lot mixed with fruit juices so as to assist dissolution. Fluoridation he regards as 'an insufficient method of adding tooth nutrients'. It is not proposed to discuss his views[8, 9, 10, 11, 12, 13] in detail. These, while stressing the nutritional aspects of tooth formation and protection, are in some respects extreme and in others unacceptable. The purpose of referring to him here is that in one of his papers[9] he claimed that the Swedish Medical Research Council had assigned him the task of drawing up a plan for testing his theories. I was concerned to inquire whether such a test had been carried out. The answer supplied by the Swedish Embassy is that his requests had not been accepted for the reason stated that he 'is not regarded as an expert scientist on teeth by specialists in this field'.

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CHAPTER 46. PALATABILITY, TASTE AND SMELL

794. None of these are affected by the addition of fluoride ion at a concentration of 1 ppm[1]. Allegations to the contrary were not advanced on an informed level and those that were made would seem to have been derived from an overactive imagination or from confusion with the effects of other substances or bacteria naturally occurring in or added to the water. Examples were chlorine, compressed air and the 'iron bacteria'. The latter are bacteria-like organisms which have the power of abstracting iron (and some, manganese) from pipes and water and which if allowed to multiply under favourable conditions of pH and organic matter cause deterioration in the quality of the water supply affecting all matters referred to in the heading of this chapter. Their presence is not an uncommon problem of water management[2, 3].

795. Tests have been made to determine a level at which some effect on taste may be expected. Water supplies of course vary in regard to their content and outside of a laboratory and doubtfully within it there is no such thing as pure water in the absolute sense. International standards for drinking water[4] permit up to 1,500 ppm of total dissolved solids in drinking water and many of these do affect potability such as iron (50 ppm), manganese (5 ppm), zinc (1.5 ppm), copper (1.5 ppm) and magnesium (1,000 ppm). The figures in brackets are the maximum allowable in each case. Further in the course of recognised water treatment procedures quite a variety of chemicals may be added either to clarify or purify the supply. Chlorine and alum are familiar examples. Residuals from these may have an effect.

796. These matters make the testing of taste difficult as distilled water as a test medium is not an exact substitute for water in which there may be anything from 100 to 1,500 ppm of dissolved solids. However, using distilled water Cox describes an experiment[5] in which it was found that 50% of 280 subjects tested failed to distinguish any difference between distilled water and distilled water fluoridated at 133 ppm. In another experiment Cox and Nathans[6] working with 187 male dental students, found only eight who could detect 10 ppm fluoride in distilled water and one only of the 187 could detect it at 2.4 ppm. It was concluded that a fluoride ion concentration of 1.8 ppm would be tasteless in distilled water and that the average person would be unable to detect any taste at 10 ppm. It is suggested that the figures would be even higher in normal (i.e., undistilled) water.

797. I may be permitted a postscript. The chlorination of drinking water is now generally accepted as a necessary measure. I claim no analogy between the purposes which it fulfils and those served by fluoridation. But when first introduced it was bitterly assailed. The following quotation from a medical paper written in 1927 seemed to me to add some perspective not only to chlorination but to fluoridation:—

> 'Since drinking water has been chlorinated, there has been a notable increase in endemic goiter, cancer, rheumatoid arthritis, neuritis, and other diseases that the predisposition, if not the actual cause, of some of these diseases is due to changes produced in the drinking water by chlorination.'[7].

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CHAPTER 47. AGRICULTURE, ANIMAL HUSBANDRY AND HORTICULTURE

798. No direct evidence was submitted alleging any possibility of ill effects from fluoridation at 1 ppm. Water fluoridated at that level is well within the tolerance levels of all animal species mentioned in the literature and is said to be beneficial to bone and teeth structure[1]. Specifically when drunk by dairy cattle it does not result in any elevation of fluoride content in the milk beyond that normally found in unfluoridated areas. It follows that other dairy products are unaffected[2]. Harvey (1952)[9] found no appreciable accumulation of fluoride in any edible portion of Queensland sheep which had been for two years on water containing 10 ppm F. Hence there is no danger to humans in the consumption of such animals[9].

799. Because of the universal presence of fluoride in water and soils, some amounts of the element can be found in all plants. Uptake and tolerance is very variable between species. A great amount of experimental work reviewed by Thomas and Alther[3] shows no indication of harm to seedlings even of susceptible species at any concentration of nutritional fluid less than 10 ppm. There is no evidence that water at 1 ppm will harm vegetables, flowers or other plants[4]. Residents of Hobart may be relieved to learn that the chrysanthemum is regarded as a resistant species[8].

800. Fluorosis in animals and plants where it does occur is generally due to airborne contamination resulting from industrial pollution. In the case of plants it is generally due to the absorption of gaseous fluoride (e.g., HF) and in the case of animals from eating contaminated foliage. The subject has been extensively studied and there is a voluminous literature[3, 5, 6, 1]. If such hazards exist and I have no evidence that they have been demonstrated in this State, they would not be significantly augmented by water fluoridation and the remedy is to control the source.

801. Another allegation which was pursued was that residues of fluoride may be taken up from fruit and vegetables which had been sprayed with fluoride-containing insecticides or other sprays. I am informed by the Chairman of the Pesticides Board that none of the pesticides registered in this State for application to fruits, vegetables or crops destined for human or animal food or for application to other plants or trees or for sheep or cattle dips contain fluorine[7].

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CHAPTER 48. COOKING AND FOOD PROCESSING

Section (a)—Cooking

802. The fear was expressed that the fluoride content of water used in cooking may by long boiling be concentrated so as to reach higher and more dangerous levels than 1 ppm. It is true that when water containing fluoride is boiled, the fluoride remains and is not diminished by evaporation. As the water content falls the fluoride level may be expected to rise proportionately but the total fluoride content would nevertheless remain the same. Thus to quote from the example used in the South African Report[1] one gallon of water at 1 ppm contains 4.5 mg of fluoride and if evaporated to one pint the concentration will increase to 8 ppm (Cf Bartlett) but the total amount present will still be 4.5 mg. As they go on to remark:—

> 'An occasional intake of food or beverages prepared from such water will do no harm and regular preparation by such a procedure is hardly conceivable—it would mean using one gallon of water to make three cups of tea'.

803. The rise in concentration is not limitless but would take place until the solubility product of some metallic fluoride (e.g., calcium fluoride) is exceeded when precipitation would occur. Beyond this point further boiling would decrease the soluble fluoride content, not increase it. However, before that stage is reached, relatively high concentrations of fluoride would certainly be achieved.

804. The subject has been investigated by Fremlin and Mathieson (1967)[2]. The importance of these investigations is that they were conducted over a range of naturally and artificially fluoridated waters, both soft and hard. They found higher levels of concentration than would be indicated by the application of simple solubility theory, but, and this is really the important point:—

> '. . . the artificially fluoridated waters behave in a similar way to the waters containing fluoride naturally so that, as pointed out by Godber (1963) the conclusions drawn from clinical observations on the latter continue to hold for the former. The recommendation that 1 ppm of fluoride be added to drinking water in temperate climates to give maximum freedom from visible defect, and a caries susceptibility reduced to near the minimum value, has been based on results from natural areas where people may do such things as leaving the kettle simmering; boiling cabbage and then using the water to make gravy or topping up the kettle for early morning tea. Similar activities in the artificially fluoridated areas will therefore lead to similarly harmless results. It is also worth noting that the behaviour of water of either kind is just what would be expected on any theory if it is boiled down only three or four times and that it is hardly credible that even this factor could be consistently reached in a normal household.
>
> 'To achieve even an average factor of 3 in a family of four using only 3 pints of drinking water each in various forms (i.e., including the water they leave in their glasses and the tea they leave in the pot, etc.) would require the evaporation of 24 pints a day, year in year out. It is improbable that 3 gal. extra of water would be carried to the stove each day just to boil away.'[2].

Nothing in the evidence persuades me that I should doubt their conclusions.

805. It was also suggested that apart from the concentration of fluoride by boiling and evaporation, in the processes of cooking and canning, vegetables and other foods would take up extra fluoride from the water in which they were cooked. Fluoride is absorbed in cooked foods[3, 4] but the total amount absorbed can be no more than what was present in the water. Whether it all becomes physiologically available may be doubted unless ingested as fluid, in which case it contributes to the total fluid intake. But the answer is as before. Epidemiological studies of communities using fluoridated water at levels much higher than 1 ppm and who presumably employ the same or similar culinary habits have not shown any untoward results.

806. Soup was another matter mentioned. The South African Commission considered that the 'Fluorine in bone is not extracted during cooking and a high fluoride content in soup cannot be expected from this source'[1] and the evidence supports this view. The only other thing mentioned was baking powder. Some powders of this class are derived from phosphate compounds and may have a high fluoride content[5, 6]. I have no evidence which would justify me in suggesting any community danger could arise from the quantities of these substances customarily used in cooking.

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Section (b)—Food Processing

807. Some references were made to possible deleterious effects that might be suffered by food processing industries if compelled to use fluoridated water in their operations. Though perhaps outside the strict scope of my inquiry, I thought it so closely related thereto as to warrant attention.

808. Inquiries reduced the matter to a very small compass. A number of major Tasmanian processors were written to direct and asked for their reactions. In addition all municipalities were circularised and asked to advise whether they had any knowledge of any problems that could arise in relation to industrial users of municipal water supplies if fluoridation were introduced. One processor only who is obliged to concentrate by evaporation fluids used in his manufacturing process to approximately 1/14th of their former volume would find the process objectionable. There would be no danger in the use of any normal quantity of the highly-concentrated product[1], the fluoride content of which would be less than a tin of fish, which may contain up to 25 ppm[2], but it is believed the product might be objectionable in the light of manufacturing and pharmaceutical specifications which at present control sales. In a plant using water on this scale, if a separate supply is not practicable, defluoridation equipment is feasible.

809. The effect of fluoridated water on such processes as baking and brewing has been specifically studied. It has been found that brewing yeasts are not inhibited by water fluoridated at concentrations up to 25 ppm and in baking processes no effects were noted up to 10 ppm[2, 3]. Its use in vegetable growing has been studied in Holland with reassuring results[4]. In short all inquiries made both by this Commission and other bodies of inquiry[5, 6] have not found any evidence of interference by fluoridated water with normal industrial processes including those involved in the processing of food[7]. Processes of course which would normally use distilled water, such as the making of pharmaceutical preparations, would be unaffected.

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CHAPTER 49. MATTERS OF WATER ENGINEERING

Section (a)—Whether There is any Danger of Overdosage

810. Some objections have been urged based on alleged difficulties or dangers in the practice of fluoridation. I have inquired into these objections and the practical matters which are involved at some length. In the outcome I think they are groundless.

811. All aspects of water treatment are skilled matters calling for expert design and supervision. Fluoridation entails problems no different in kind and its practice is within the normal competence and skills of water engineers and chemists[1, 2]. I do not recommend that it be put into operation except under expert guidance, but once installed and working to design standards, its day to day operation is within the competence of skilled tradesmen so long as professional supervision is maintained and technical maintenance of equipment is available when and where necessary.

812. Fluoridation has been practised for twenty-five years. A range of equipment is available. It is fully described in the literature[1, 3, 4,5] and it ranges from simple solution feeders, the input of which is metered according to the quantity or flow of the water to be fluoridated, to sophisticated volumetric (dry) feeders electronically controlled to deliver a measured amount of chemical to solution tanks the level and concentration of which are automatically controlled to correspond with the flow of the raw water to be treated. They are available in Australia and Tasmanian experience is adequate for their installation, maintenance and operation. While in general the solution feeders seem to be more suitable for smaller installations[6] the choice of equipment for any particular water supply is a matter of engineering design depending on the form of fluoride supplement, the volume of water to be treated, whether other forms of water treatment are to be undertaken as well and the general layout of the waterworks.

813. Fail safe devices are universally employed, i.e. the equipment is so designed as to cease to function should there be any irregularity in power or water supply or in the functioning of regulatory devices such as meters. Other inbuilt safety factors against overdosage are incorporated in design principles relating to hopper and solution tank sizes. Particularly in the case of sodium silico fluoride the designed size of solution tanks is generally insufficient to permit dissolution of any abnormal quantity of solid chemical and this safety factor can be coupled with other devices which shut down the fluoride supply immediately any undissolved solids are detected in the solution[6, 7].

814. It is standard practice for the water operator daily to test by simple colorometric analysis the treated as against the raw water at the works. Similar tests are also made of water samples collected from delivery points in the system. Periodical samples are forwarded to the Government Analyst for comparison analyses by more sophisticated analytical techniques. There is a further check available in the simple accounting for the solid chemical used in relation to water supplied. These are and should be matters of standard procedure laid down and enforced by the appropriate health authority. The cumulation of all these measures is such that even allowing for error or neglect the possibility of any of the horrendous results pictured by some witnesses as a result of accidental or mechanical gross overdosage is so remote as not to call for further consideration. Some of the relevant logistical facts have been mentioned previously (Ch. 18, *supra*).

815. However apart from overdosage gross enough to cause immediate symptoms of toxicity, the possibility of slight but constant overdosage has been raised by some. These objections stem I think from unfamiliarity with the practical working of these schemes. The object of the daily analyses is to test by actual result the calibration of the metering devices or feed regulators. The machines are of course capable of instant manual adjustment. The operator's accuracy is checked by periodical check analyses made in the laboratory of the Government Analyst. The amount of chemical used and that remaining at any given time in relation to gallons of water delivered is a simple proportion sum which in the more sophisticated machines is portrayed graphically. In any case it is the practice for the operator himself to keep it under surveillance at the works, to record it and for his returns in relation thereto to be checked again by a higher source.

816. As an example of the very close relationship which can be obtained between these different methods of checking, Table XXXIII from Longwell (1963)[6] summarises the results for the first five years operation of the three test fluoridation areas set up as a result of the report of the U.K. Mission in 1957. In his figures 'calculated' means the proportion which the gross amount of chemical used bears to the metered water output of the plant; 'determined' means ascertained by actual analysis.

TABLE XXXIII

DAILY CONCENTRATION OF FLUORIDE ION IN THE WATER
(*a*) *Calculated* (*b*) *Determined*

	Number of Samples	*Mean concentration, ppm F. (brackets show standard deviation)*	
		(*a*) *calculated**	(*b*) *determined*
Anglesey (Gwalchmai) 17 May 1956 to 31 May 1961—		0.90 (0.11)	
Plant tap	1,773	....	0.98 (0.08)
Distribution system	3,215	....	0.97 (0.09)
Check analyses at L.G.C.	243	....	....
Kilmarnock 25 April 1956 to 31 May 1961—		0.95 (0.13)	
Plant tap	3,590	....	0.94 (0.10)
Distribution system	3,147	....	0.96 (0.10)
Check analyses at L.G.C.	327	....	0.92 (0.12)
Watford 1 February 1957 to 31 May 1961—		0.95 (0.10)	....
Plant tap	3,272	....	0.95 (0.07)
Distribution system	5,813	....	0.92 (0.07)
Check analyses at L.G.C.	447	....	0.92 (0.10)

* In the calculated figures no account has been taken of fluoride naturally present which can vary from a mere trace to 0.1 ppm.

Commenting on this Longwell said[6]:—

> 'It is clear from Table XXXIII that the desired concentration has been maintained consistently at all three study areas and moreover, that the calculated concentrations agree very closely with those chemically determined. The check analyses carried out at the L.G.C. using rather more precise techniques were in close agreement with those carried out by the plant operators and showed that the chemically unqualified staff at the waterworks had worked carefully and efficiently.'

817. The object is a mean at the optimum level. Day to day variations either side of the mean are to be expected, but their effect should not be overstated. A variation of 10% either way is equivalent to one ten millionth part of fluoride, the range of difference being 0.90 to 1.1 ppm. Physiologically such differences are meaningless.

818. In practice no difficulty has been experienced in achieving tolerances of 0.1 ppm and most installations achieve less[1, 3, 4, 6, 8]. In Hobart it is claimed the tolerance of the manual control by which the operator can make any adjustment required is half that figure, i.e. 0.05 ppm[9].

819. So much for possible overdosage. I am satisfied that the fears expressed in relation thereto are groundless. What has happened however in Tasmania is that underdosage has been common, and not only in the initial stages of fluoridation. The result has been in Hobart and Beaconsfield (I do not include Launceston) that the optimum level, so far from being exceeded, has not even been attained by a margin substantial enough to call for comment.

820. Several factors have been identified; teething troubles with unfamiliar plant and equipment, delay in provision of spare parts, under-design (machine incapable of handling water bypassed into system to cope with peak demands), electrical failures (e.g. for a prolonged period following the 1967 bush fires) and in Beaconsfield a failure to provide for a relief operator (during his necessary absences on leave or through sickness the plant was not working). These are contributing causes, the dominant cause, I am satisfied, has been a lack of confidence in public backing, resulting in a general desire on the part of all concerned to lean over backwards so as not to provoke public opinion by any circumstances which might be employed to found an allegation of overdosage. Hence underdosage has been the aim, rather than a mean which necessarily involves slight variations either side of the optimum. The quantities delivered for Hobart and Beaconsfield are as set out in Figure III, Ch. 17 (c) (*supra*).

821. These deficiencies are avoidable, and there is no engineering problem involved that cannot easily be overcome, though there is a reluctance to incur any expense involved in doing so before the result of this inquiry be known.

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Section (b)—Whether There is any Danger of a Build-up of Fluoride in the Reticulation System

822. Another fear expressed postulated an unpredictable build-up of fluoride within the reticulation system resulting either in water being discharged at some outlets with concentrations greater than the optimum level or in the discharge of sludge or rust products with heavy fluoride concentrations. I have had no satisfactory evidence in support of these hypotheses and they are supported by no engineer or chemist who appeared before me except theoretically to the following extent.

823. It is not understood how more fluoride can come out of a given system than that which is put in in the absence of some form of precipitation and redissolution or distribution in a solid form. On the other hand it is far more feasible and perhaps to be expected that the level at outlet will be very slightly less than the concentration at the inlet point. It seems that this is particularly likely to occur in the early stages of fluoridation[1], due to the dilution of fluoridated water with unfluoridated residues in mains and reservoirs but also by the longer process involved in the precipitation and absorption of fluoride in relatively insoluble combination with other salts in the tubercles or corrosion products that form inside the pipes. The fluoride concentration possible in such products is limited by the precipitation factor and solubility of the compound formed. At 1 ppm of fluoride this would require a considerable concentration of calcium or magnesium to mention two of the salts most likely to be implicated for reasons discussed elsewhere. Chs. 40 and 41). In fact in the opinion of Longwell[2, 3] it would not occur in ordinary water and this opinion would seem to be confirmed by the San Francisco analyses discussed below[4], and by the recorded results which consistently fail to record any but trifling differences between levels at different points over extensive reticulation systems[2, 5]. Any problem of this nature that was likely to be encountered would be first encountered in the solution tanks where the concentration of the fluoride is much higher than in the distribution system. The U.K. Report of the Research Committee of the British Ministry of Health[6] shows that it can easily be avoided.

824. It is further the nature of such water pipe tubercles firmly to adhere to the inner lining of the pipe and to resist removal except by forcible means. 'Sludge', not defined (but I take it some form of corrosion product was indicated), was also mentioned, the suggestion being, as with the tubercles, that an unwary consumer may drink such a quantity of solid or particulate matter as to result in an overdose of combined fluoride.

825. Many matters are here confused, some of which have been dealt with elsewhere, but as to the general question I quote from a definitive study[5] conducted by the New York University College of Engineering into the Water Fluoridation Practices in twenty major cities of the United States, five of them serving populations of one million or more.

> 'Neither the length of line, time of flow, age of pipe, nor materials to which water is exposed affect average fluoridation content in distribution systems significantly.
>
> '. . . Special studies of F ion residual along routes of flow have shown only variations that might be anticipated as a result of the accepted practices and within the reasonable limits of error of sample collection and analysis. In no instance is pipe line corrosion traceable to fluoride content reported.
>
> '2. These records show no evidence of feedback of concentrations of F ion from such postulated sources as pipe coatings, tuberculation, treatment sludges, storage reservoir sediments and the like. These studies have shown on the contrary that to achieve undesirable F ion concentrations by such addition would require a quantity in suspension of the coating, sludge or rust so great as to render the water non-potable and unfit for use because of its presence. In one example of hydrant flushings with water clearly unacceptable for normal use, the maximum F ion content of the supernatant liquor was 1.32 ppm and below the maximum suggested by the Public Health Service'.

The South African report may also be consulted[7].

826. The expert evidence which I have taken accords with the views cited. The New York study should be referred to on all matters discussed in this chapter. Perhaps I should mention one small qualification arising from the evidence. Two of a number of samples of Beaconsfield water taken by an officer of the Commission gave a value of 1.19 and 1.26 ppm F^-. Inquiries made revealed that one sample had been taken from a tap in a vacant building which had been locked and not used for six months and the other was taken from a garden tap of a vacant dwelling, in both cases it was the first water issuing from the tap, i.e. no water was run off before the sample was taken. The explanation given by the Beaconsfield water chemist confirmed by the Government Analyst, I think, acceptable, viz., that evaporation over a period of time at the mouth of a slow dripping tap would leave a deposit of fluoride and this, on being flushed from the tap mouth, would result in a concentration in the first water issuing higher than normal. The amount as distinct from the level or concentration in such a sample would be negligible. All other samples taken (seventeen in all) on the same occasion from both vacant and occupied premises were from 0.88 to 0.99 ppm.

827. I was also informed of a reported analysis of a sample of Yass (N.S.W.) water which allegedly gave a figure of 4.75 ppm although the water is fluoridated at 1 ppm. A lot of time was spent following this report and after considerable difficulty, in the closing stages of the Commission the person who took the samples was finally traced in another State. The sample was allegedly taken without witnesses from a garden tap in the Municipality nearly ten years ago, and sent to a private analyst who returned a value of 4.75 ppm. I have not been informed of the method of analysis. I have perused the records of the Yass water plant and can obtain no corroboration or explanation if the report be accurate. It could be that the explanation is on the lines suggested in respect of Beaconsfield, but I am not sufficiently impressed by the circumstances to cause me to doubt the accuracy of the conclusions already expressed.

828. At the same time it is my opinion that the system of sampling and check analyses of Tasmanian fluoridated systems as carried out in the past has weaknesses. Testing points have been allowed to be standardised and would seem to have been chosen for their convenience rather than as a purposeful attempt to survey the whole system being treated over a period of time[6]. Bulked samples, the effect of which must be to mask deviations in individual samples, have been customarily employed.

829. It should be remembered in the words of Longwell[2]:—

> ' The fluoridation of water supplies is a public health measure, and it is important that the required concentration of fluoride in the water be maintained accurately without daily or even hourly fluctuations, and proof of this should be available '.

Methods such as I have described do not supply the objective standard of proof that the status of fluoridation as a controversial issue of public health demands. Daily samples taken from the same fixed point every day and then bulked for a month before being sent for check testing will not instil confidence in a nervous public that deviations in concentration could not occur and remain undetected. Scientifically even if they did the result may be negligible but public confidence will not be enhanced by such methods of supervision.

830. Sampling and testing routines should be reviewed so as to fulfil the highest criteria demanded by a critical public opinion and full compliance should be insisted upon. Perusal of the records kept shows that while the preoccupation has obviously been to exclude the possibility of overdosage at the waterworks, underdosage which has in fact occurred does not seem to have been seriously regarded. While this, as I have remarked, no doubt reflects the lack of confidence felt by those responsible in public backing, if fluoridation is decided upon more care should be taken to ensure not only that the optimum level is not exceeded but that it is maintained. This should be done not only for clinical and epidemiological reasons, but as a means of retaining public confidence in the administration of fluoridation schemes generally. There is some point in the objection that ' it is not worth having if we don't get what we pay for '.

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Section (c)—Alleged Corrosive Properties of Fluoridated Water

831. It was also urged that fluoridation either caused or aggravated corrosion in pipes and other appliances. Tasmanian experience[1, 2, 3] (Beaconsfield 15 years, Hobart 3 years) directly contradicts this assertion and it has no scientific backing[4, 5, 6, 7]. The matter was discussed fully with Professor O'Donnell, with a consultant water engineer and with other engineers responsible for the maintenance and oversight of water systems carrying fluoridated water. Nothing emerged from their combined knowledge or experience to give any support whatever to a corrosion hypothesis.

832. As against this completely satisfying evidence both theoretical and practical my attention has been drawn to statements and other documents mainly from America (but some from N.S.W.) alleging extensive corrosion in water supply systems and household appliances as a result of fluoridation. Such statements as I have been able to investigate do not stand up to critical examination. One example will suffice.

833. Ex. 103[8] is a notarially certified statement (undated) by Willard E. Edwards described as a 'Registered Professional Engineer and Corrosion Consultant'. I extract the following:—

> 'San Francisco has had frequent instances of burst water mains following fluoridation. One 16-inch main burst with a resulting repair damage and clean-up cost of $30,000. Pipe sludge and laboratory analyses show a fluoride content. Reliable analyses in Los Angeles showed a fluorine content in sludge at the breaks of from 1100 to as high as 22,000 ppm',

and private analysts' certificates[9] in support are attached.

834. The comments of the San Francisco Water Department have been obtained and are contained in various exhibits. They are too long to quote in full, but the following extracts should be sufficient.

As of 9 August 1963[10]:—

> 'The San Francisco Water Dept. has been fluoridating since 1952 and have never been able to find any evidence of corrosion in its distribution or transmission system due to the addition of fluoride'.

As of 26 December 1967[11]:—

> 'Investigations of piping systems have shown no corrosion which could be attributed to fluoride nor has there been found any evidence of build-up in the water system.
>
> 'The investigations were made by analyses of bottom deposits in distribution reservoirs and pipelines and also by entering our sixty (60) inch transmission lines. Visual observation shows no build-up and tests of scrapings also show no build-up of fluoride . . .'.

As of 29 April 1964[12]:—

> 'We here in San Francisco have also seen published data as referring to analysis made on pipe from this break and the sediment therein with results in thousands of parts per million. In attempting to ascertain where or how the laboratory that performed these experiments found results in the thousands of parts per million, we made numerous analyses changing the methods of sample preparation and analytical methods, it was found that by poor grinding and certain techniques in acidic distillation, coupled with interferences that are normally in this type of material, we could reproduce results which were very erratic and showed high fluoride levels, but upon refining our techniques and comparing these against the other laboratories we found consistent rather low results in fluorides. In order to confirm this data the San Francisco Water Department Laboratory, the San Francisco Health Department Laboratory and the California State Department of Public Health Laboratory all ran analyses on the same samples taking aliquot portions. Results were as follows: (Results in milligrams of fluoride per kilogram of sample—parts per million):—

Sample	S.F. Water Dept.	S.F. Health Dept.	State Health Dept.	Average
'1. Reservoir Shoreline Mud (no fluoride added)	180	167	140	162
'2. Outer incrustation of pipeline	200	201	180	194
'3. Bottom Mud Sunset Reservoir (fluorine added)	240	214	140	198
'4. New pipe lining (never in service)	90	100	80	90
'5. Old pipe lining (from break at 34th Avenue and Yorba—fluoride added)	100	105	80	95

'It is also interesting to note that no person asked this Department for permission to take samples from the break referred to, nor have we been able to find any Water Department employee who was aware that a sample from this break was collected, although Department personnel were working on the break night and day to get the job done. The results of these analyses show that a person would have to chew and swallow more than one pound of concrete or muddy rust to merely ingest 0.0035 ounces (100 milligrams) of fluoride ion, and this would happen if the water was fluoridated or not.'.

835. Another much quoted allegation in relation to the City of Wilmington, U.S.A., was not sustained by inquiries made of official sources[13]. Similar highly-coloured allegations were made to the New Zealand Commission relating to the American cities of Northampton, Detroit and Newburgh. Inquiries proved all of them equally groundless[14].

836. One wonders as did the New Zealand Commission about the *bona fides* of these reports. Assuming they are genuine the most probable explanation would seem to be that they spring from a failure on the part of some analysts to appreciate the difficulties of analysing solids such as water pipe tubercles for fluoride content. Such analyses are extremely difficult and pose quite different problems from analyses of solutions. I was at one time informed that it was doubtful whether they could be performed in Tasmania.

837. Other comments could be made and have been made in the evidence[15]. I will take as an example and assume its accuracy a cited figure of 3.9% (expressed as NaF) in respect of the solid scrapings from an alleged corroded washing machine. The nature of the other 96% of the solid is not stated, but assuming it to be either calcium or magnesium, for these salts to have precipitated fluoride out of water fluoridated at 1 ppm would require such a concentration of them that one wonders how the water could be used for washing at all. This comment is reinforced by the Report of the Department of Public Health, N.S.W., to whom this report was referred. I quote a paragraph[16]:—

'In all, eleven dealers and washing machine mechanics were interviewed and all stated that the number of washing machines brought in for repair had not increased since the introduction of fluoridated water in the towns in question. The consensus of opinion was that cheap washing powders and the hardness of water brought about by the drought had been the main causes of the small amount of corrosion experienced. One interesting statement by a dealer at Condobolin was that most of the machines affected by corrosion were from rural areas which are not supplied with fluoridated water. This was borne out by a maintenance mechanic at Orange who stated that of 100 washing machines repaired during the previous year, only two were affected by corrosion, and both were from areas not supplied with fluoridated water'.

838. It would be tedious to go through other allegations of the same quality. Other complaints[17, 18] of corrosion or damage to household appliances have been referred for inquiries where possible and discussed fully in the evidence. In no case have they gained any substance as a result.

839. One constantly occurring assumption for which I understand there is no foundation is that if fluoride is found in combination with scale or corrosion products that it is the fluoride that is responsible and not the other salts to which the fluoride has been attracted and with which it has combined[15]. In point of fact calcium fluoride which is the anticipated form in which fluoride so combined would be found[7,15] is not corrosive at all. A related suggestion[8] alleging a destructive electrolytic action for water fluoridated at 1 ppm was described by Professor O'Donnell as based on chemically indefensible premises[4].

840. I might also mention that a circular letter sent to some 17 N.S.W. Municipalities practising fluoridation failed to bring to light any engineering problems of any significance or any allegation of corrosion in the reticulation[19].

841. As to corrosion generally I can only report my firm conviction in relation to fluoridation at 1 ppm this is yet another of those hares which when started up and pursued turn out to be nothing more than a small fish of crimson hue.

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CHAPTER 50. THAT FLUORIDATION IS WASTEFUL

842. A common objection to fluoridation is that it is wasteful in that only a very small portion of the total fluoridated water supply is drunk by those whom it is intended to benefit. Inasmuch as the greater part of communal water supplies is used for other purposes, e.g., for industry, sanitation, horticulture and bathing, the argument is that it is an extravagant and wasteful method of achieving a limited object.

843. The premise is correct, the conclusion, I suggest, is wrong. It cannot be disputed that the greater part of water that might be supplied in any Tasmanian community would be used for other than drinking or dietary purposes. An exact ratio is difficult to arrive at for obvious reasons. I have had estimates ranging from 90%[1] where there is a substantial industrial use to 66⅔%[2] where there is none. I see no reason to question them. It is also plain that the fluoride added to this water—the bulk of it—which is not drunk or used in the preparation of food does not fulfil the purpose for which it was added and is in this sense wasted.

844. The same of course is true of chlorine where used and perhaps of other water additives as well. It is still cheaper to fluoridate or chlorinate the whole of the water supply than to attempt to differentiate the supply according to the purpose for which it may be intended.

845. I have been furnished with calculations designed to show that it is cheaper to supply every child in the community with a free yearly supply of tablets than to fluoridate the whole of the water supply[3]. The estimates are arguable, but, even if they are admitted, the relative inefficiency of tablets as a public health measure is so much greater that I do not think the comparison is worth pursuing. Detailed reasons are given in a later chapter (Ch. 52—Alternatives).

846. Relative costs are only one facet of the argument, relative efficiency is the other and the more important. One cannot be separated from the other and the economics of the matter cannot be determined without considering the intrinsic worth of what you pay for. When both aspects are taken into account water fluoridation remains the better, i.e., the more effective means of achieving the end desired.

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PART K—TERMS OF REFERENCE—QUESTION 3

'Whether it is practicable and desirable to leave the decision whether or not to add fluoride to water supplied to the public to local or other public authorities responsible for such supply and if not to whom should such decision be entrusted?'

CHAPTER 51. BY WHOM DECISION MADE—PARLIAMENT—LOCAL GOVERNMENT—REFERENDUM

847. This question has posed difficulties. I have interpreted it broadly so as to require from me an unmistakable recommendation as to the political level at which the decision whether to fluoridate or not should be taken. To construe it more narrowly would, I feel, leave unanswered some of the major matters that underlie the question and which have been debated at length before me. I am conscious that a Royal Commissioner is unhappily placed in dealing with matters which involve both political decision and constitutional responsibility. Factors which are political in their content merely are not capable of assessment in an inquiry of this sort nor is it part of my function to say whether they should or should not be allowed to modify recommendations which I make based on technical considerations of benefit to be achieved and efficacy in its provision. In what follows I seek to place considerations of the latter kind in a traditional context of administrative and constitutional responsibility.

848. Whether or not to fluoridate requires a decision on a major matter of public health. 'Public health' as a science or art describes those measures for the promotion of physical well being and the prevention and control of disease which are or should be taken by the State in the general interests of the community either because their importance is such that they should not be left to individual responsibility or because their effective implementation is beyond individual capacity. In a political context 'public health' implies both the power and the duty of a lawfully constituted Government to take such measures as the considerations stated call for[1]. Traditionally the duty to do so has been emphasised and regarded as more cogent where the problem of health involves the young and the impotent. Under all these heads and on the additional ground of its technical sophistication fluoridation qualifies as a major matter of public health.

849. As such, the decision should be one of Government taken in and by the lawfully constituted repository of sovereignty in the State which is Parliament. To me the question asked admits of no other answer. In a unitary constitution founded on traditional principles of British constitutional law, Parliamentary sovereignty is unique and indivisible; whatever be the responsibilities that may be kept in mind in its exercise, it—sovereignty—is shared with no other body not even the electorate[2].

850. These are general considerations that underlie the remarks that follow. In them I seek to reflect the evidence I have taken and to relate its content particularly to considerations of Local Government in this State. The major cities of the State have already fluoridated and there is nothing to suggest any corporate uncertainty as to their position. As a result, in what follows, I am perhaps more conscious of the difficulties that have been encountered at the municipal rather than the metropolitan level. But I draw no real distinction. Such differences as may be suggested between metropolitan and municipal competence to deal with the matter of fluoridation are quantitative, i.e. they are matters of degree only and would not have affected any recommendation that I make.

851. Fluoridation has both scientific and what I have elsewhere called (Chapter (44)) sociopolitical implications. From both aspects it transcends the capacity and the role of Local Government as it exists in this State satisfactorily and competently to arrive at a decision. Technically Local Government is not competent to resolve the medical and dental issues involved. Few Councils, if any, have a full time medical officer. None have full time dental officers. They have not, even in those municipalities where the part-time services of such an officer is available, the benefit of direct recourse to the specialist and wide ranging advice of a central Department of Health which has technical resources in personnel, skills, literature and research material. Awareness of such a lack has been reflected in the evidence. Minutes have been perused revealing much uncertainty and understandable hesitation. They reflect not vacillation but a genuine difficulty felt in the decision of a technical issue too often obscured by emotionalism and by intimidatory propaganda masquerading as science. One example of many will be found in Resolution 15686 of the Devonport Council dated 27 August 1963. The work there referred to[3] is dis-

cussed at some length and dismissed in Chapter 32 (b) of this Report. Men who feel keenly and who wish to live up to the responsibilities of their position, say accurately enough, they are not competent to decide. Most councillors who gave evidence regarded it as I do as a matter for Parliament.

852. Administratively, Local Government has not the resources or the power to conduct the far-reaching inquiries which the subject may be thought to require. Governments may appoint Royal Commissions or other bodies of inquiry, Houses of Parliament may appoint Select or Joint Committees. They have powers in relation to the compulsion of witnesses and the production of documents not possessed by Municipalities. They have access to a range of technical advisers not available at lower levels.

853. However, the matter not only calls for technical appraisal at a superior level, but as has been indicated in Chapter 44 of this Report it also involves other questions of importance. Whatever be the opinion as to whether fluoridation will confer dental benefits or otherwise, these important questions require a decision that can only be taken at the political level. The matter is not one for specialists or experts until the political decision has been made by the proper constitutional authority.

854. Fluoridation in importance is comparable to major public health decisions of the past. It has I think unique features and though I deprecate any attempt to attribute any pejorative sense to a conventional label it does possess implications in regard to both the present and the future which entitle it to be regarded as a precedent. These considerations place it in my view beyond the scope of Local Government authority, and no responsible person in evidence has suggested that they are otherwise.

855. Four further considerations are relevant. They are drawn from other portions of my Report. Any implementation of fluoridation must come more and more to be dependent on the regionalisation of water supplies in this State which from the evidence plainly emerges as the future pattern. The difficulties of a central water authority seeking to serve conflicting municipal wishes in regard to fluoridation from a common supply are obvious. In some cases I would be doubtful if fluoridation costs could be kept to a reasonable figure if regionalisation is not undertaken. Second is the need, if fluoridation is introduced, for professional and technical supervision by a central body and for the continuous surveillance that the matter calls for in relation to industry, medicine, pharmacy, and public health. Thirdly fluoridation in practice calls for close integration with the services of the Dental Division of the Public Health Department, particularly that of the Schools Service. Lastly the problem of caries is not local or regional, urban or rural, but State wide. It is not determined or limited by boundaries or residence.

856. Fluoridation is already the direct concern of a major State Department and there is no comparable municipal authority with the same responsibility, knowledge or skills. I express the firm conviction that it is not really a problem of water supply at all but of health; water supply as the medium or vehicle of administration is but an accident not capable of changing the essential character of the matter proposed as a public health measure. '. . . . it is not to promote the ordinary use of water as a physical requisite for the body that fluoridation is proposed. That process has a distinct and different purpose; it is not a means to an end of wholesome water for waters function but to an end of a special health purpose for which a water supply is made use of as a means.'[13]. If fluoridation cannot be justified as a matter of public health it cannot be justified at all, and that mode of reasoning which ascribes to it the character of a Municipal responsibility because water supply is involved, is, I suggest in error.

857. All these considerations point unmistakably in my finding and recommendation to the conclusion that the primary decision is one for the ultimate repository of constitutional authority, i.e. the Parliament. To be explicit I think Parliament should say, not as it has done, that Municipalities may fluoridate if they wish which is but to avoid the question and not to resolve it, but whether as a deliberate measure of Public Health it shall be introduced. There is in my opinion no other way satisfactorily to conclude this controversy. This does not prevent the qualification that the decision having been made it shall, if favourable, only be put into effect after technical studies have demonstrated its feasibility in relation to particular areas and the proper administrative arrangements have been made for the installation of approved equipment and its supervision. These are matters of detail in administration, not issues of policy.

858. A decision such as is here envisaged has been made by competent legislatures in Western Australia[15], in Eire[5] and in the States of Connecticut, Illinois and Minnesota[14] in the United States. A newspaper reference (Herald 20.10.67) says that several other American States are currently examining similar laws.

859. The Western Australian legislation[15] reflects generally the recommendation that I make. The matter was debated in Parliament on a Bill the object of which was unmistakable, i.e., to authorise the responsible Minister on the advice of a technically competent Committee, to direct that fluoridation be put into effect by any water supply authority. The Parliamentary issue was plain whether fluoridation as a deliberate and controlled measure of public health was to be introduced in the community and the issue was debated as such in all its aspects in both Houses[4]. The Eirean legislature faced the same issue in the same unmistakable terms with similar results[1, 5, 6].

860. The fact that at the time the control of communal water supplies in Western Australia was already vested (with one or two minor exceptions) either in a metropolitan water authority or in the State Public Works Department, though it may have simplified issues, does not seem to possess any significance. It is the nature of the issue itself not of the water authority or the means whereby it may be implemented that should determine the appropriate forum for the decision whether or not it is to come into force as a community measure.

861. It was suggested strongly both in evidence and in argument that a decision to introduce fluoridation in any given community should be preceded by and be conditional upon a referendum in the community concerned. It, a referendum, as the appropriate forum of decision was urged by the opponents of fluoridation on a number of grounds which may be resolved into the general proposition that it is the right of members of the community to accept or reject by popular vote any important matter which may be thought to affect their health or welfare. As I have already pointed out in a British Parliamentary democracy there is no such constitutional or legal right any more than there is in other matters affecting public welfare such as safety, law and order, taxation or defence. Many however were plainly motivated by the expectation that a referendum would provide an unfavourable vote against fluoridation, and I heard much evidence as to the results of referenda conducted in many parts of the world and conjectures as to the reasons therefor. In other words, the passions aroused by this issue are such that I suspect that the claim for a referendum is pursued for tactical as well as for altruistic reasons.

862. Much of the detail of such evidence is irrelevant. A general claim that opponents of fluoridation have won more referenda (when held) than they have lost is I think correct[7] and can be conceded without counting polls: a counterclaim that no referendum has ever attracted more than a minimal poll of whatever class of voters were consulted is equally I think correct without numbering heads. It is also obvious that such referenda have more often than not been conducted in an atmosphere approaching hysteria and that much (but not all) of the literature and propaganda employed (and it can be referred to by no other term) is unscientific, emotional and designed to frighten not to enlighten. I mention these matters for information. They are not in my view relevant except generally to indicate the unsuitability of the forum suggested.

863. I do not feel called upon to discuss the political theory of a referendum at length. I have referred to the fact that they have no place in the constitutional theory and structure of a unitary State in relation to matters within its sovereignty. In such matters Parliament has legislative supremacy and I would add, with respect, responsibility. Neither are shared with, though they derive from the electorate. A referendum is an innovation of Federal unions of otherwise sovereign States[8]. They no doubt reflect the uneasy union in which such Federal bodies are often conceived and do so by reposing, generally as to a limited class of matters only, ultimate sovereignty in a body which may be the electorate at large, but which at any rate is other than the Parliament or elected assembly. In Federal unions of States they serve political purposes quite removed from the exercise of constitutional powers of Government in unitary constitutions.

864. There are some minor analogies in the history of Local Government in the form of local option polls, which relate generally to expenditure on sumptuary matters or to simple matters of local administration. Such polls are provided for in an enlarged form in modern legislation in Section 186 of the Local Government Act 1962.

865. There are no analogies deriving from any of these matters applicable to weighty decisions of public health such as fluoridation. Fluoridation is a complex and technical matter. The labours of this Commission I hope will bear witness to this. It was admitted even by those who supported a referendum that such a step would necessarily involve an appeal to an uninformed electorate in the sense that many of the issues would be beyond the ability of voters to comprehend[9, 10, 11]. There was no unanimity as to whether it should be decided by compulsory or optional vote; whether it should be confined to adults or extended to minors; whether a vote should be the privilege of those who would bear the financial burden, whether as ratepayers or perhaps as taxpayers or whether it should be extended to the electorate generally and above all there was not any satisfactory answer to the argument that the interests of those most vitally interested in the result,

viz., children up to fourteen years of age, would be the most poorly represented. I think there is no profit to be gained from pursuing such matters. In my opinion they reflect merely the unsatisfactory quality[12] of a public referendum as the forum of choice in a technical and difficult matter of public health.

866. To summarise. The decision, whether or not fluoridation, as a measure of public health designed to protect the dental health of the young, should be put into force in this State, should not be left to local authorities. It is a matter for the decision of Parliament. A referendum as a means of arriving at this decision is not only without constitutional warrant but is highly unsuitable as well. It follows, consistently with the views I have expressed, that to refer the matter to a forum both technically incompetent and constitutionally incapable would constitute an abrogation of Parliament's responsibility.

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PART L—TERMS OF REFERENCE—QUESTION 4

'Whether there are any practicable methods of ensuring an adequate daily intake of fluoride by those whom it is sought to benefit other than by the addition of fluoride to water supplied to the public and, in particular, whether the supply of fluoride tablets to individual members of the public could be a satisfactory substitute'.

CHAPTER 52. ALTERNATIVES TO WATER FLUORIDATION

Section (a)—General

867. The question asked is related specifically to 'those whom it is sought to benefit', and these I take to be the children of the community as a whole and not only the children of those parents who by reason of superior fortune, education or intelligence can be relied upon voluntarily to co-operate in such means as preventive dentistry may have to offer for avoiding or at least minimising the effects of caries in their offspring. The distinction is important. There are ways other than fluoridation of either avoiding or reducing both the prevalence and severity of caries in individuals; there are none as effective or as comprehensive as fluoridation if the interests of the community are the determinant.

868. No one has suggested that fluoridation is the only means whereby the prevalence of caries may be reduced. Strict dietary control, seemingly only capable of any group application under institutional conditions, or as a result of wartime stringency has proved effective as long as the diet is persisted in (Chapter 11, Sec. (d), *supra*). Unfortunately as a community measure it fails on several counts but principally on the score of the seeming impossibility of getting the public as a whole, and school children in particular, to follow a suitable dietary regime[1]. It is also doubtful how long protection gained by this means is maintained when dietary discipline is relaxed.

869. The fluoride ion is the same however administered and will have the same physiological effect so long as it is administered in soluble form. Many methods have been suggested for its administration. They range from bread to chewing gum. There are only two worth considering, but I mention a third because it was urged by those opposing fluoridation as a preferable alternative to water fluoridation.

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Section (b)—Fluoride Tablets

870. Fluoride tablets (for which standard formulae exist) are capable of being almost completely absorbed particularly if taken between and not with meals[1], and do have the advantage that the dose is certain and fixed. Although doubts[2, 3] have been expressed whether the metabolic effect of a single tablet taken once a day will be the same as the intermittent dosage achieved by drinking water over the same period, these have not been confirmed by balance studies[4] though some regard the matter as still open[1]. Some small differences in metabolic availability have been demonstrated in other studies, but, in view of the uncertain water intake of young children it would nevertheless seem that, even allowing for those differences, in those children that take them, the dosage is more certain than could be predicted for the same group under water fluoridation[5]. However, the lack of any topical effect on erupted teeth is a disadvantage and though it may be possible to achieve a slight uptake if tablets are sucked, chewed or dissolved in water before being taken for maximum protection it would seem necessary for tablet administration to be supplemented by topical applications as indicated in paragraph 873 (*post*).

871. Supplemented in this fashion tablets are certainly effective[6] as a measure of individual prophylaxis. Indeed, I believe the effect of the evidence to be that if taken regularly and consistently from birth and if supplemented as stated they are capable of conferring a measure of caries resistance which will surpass the average statistical expectation of a child of the same age drinking fluoridated water. This is because they ensure an adequate dosage for each child who takes them, so long as they are taken. It is exemplified by the experience deposed to by Professor Martin in particular in relation to supervised programmes of tablet administration conducted by the School of Dentistry in the University of Sydney[7].

872. However, both as a continuing means of individual protection and as an effective measure of public prophylaxis, they are subject to severe limitations, so severe that they do not in my opinion rank as a feasible alternative to water fluoridation.

873. In the first place the number of years over which they need to be continuously taken every day in order to achieve the optimum benefit is still an open question. Eight years from birth is the minimum and marks the most critical period; up to 15 years would I think be regarded as preferable. Further the clinical evidence indicates that from about the sixth birthday on they need to be supplemented by topical applications, i.e., painting the teeth with strong fluoride solutions every six or twelve months to replace the uptake by ionic exchange which would occur in the alternative process of drinking fluoridated water. This is a clinical, time-consuming and costly procedure not feasible as a measure of mass control if only for the reason that there are not the trained personnel sufficient to carry out such a programme.

874. Some measure of the protection achieved will be lost to the teeth once the tablets are discontinued by back ionic exchange, and the process will be continuous[8]. The earlier in life they are discontinued the more substantial will be the loss[9, 10, 11, 12] and by analogy with water fluoridation it would seem that in children a few years' withdrawal may be sufficient for them to revert to caries rates approximating those of an unfluoridated community. It has been postulated that for maximum benefit they should continue to be taken into adult life[9].

875. The successful administration of tablets as a caries prophylaxis measure therefore calls for a degree of parental responsibility and persistence that is so high as in the mass to be regarded as unattainable and this has been confirmed by experience. It needs moreover to be supplemented by clinical procedures, which are hardly feasible when sought to be applied to the child population as a whole.

876. I am not aware of any publicly administered tablet programme that has shown any glimmer of success in relation to the vital aspect of sustained administration over even the minimum period of the first eight years of child life. Moreover, it unfortunately would seem to be true that the children in greatest need of dental assistance would be the inevitable and the worst sufferers because of the inadequacies of a programme which calls for the voluntary, informed and continuous co-operation of a conscientious parent. In other words the parents who will be most likely to live up to the full responsibilities of such a programme will be those who, even in its absence, would have sought the maximum dental care for their children. One can hardly infer the result of a public health programme from such success as tablet administration may have achieved in dentists' families.

877. Fluoride tablets free of charge have been available in a number of Tasmanian municipalities since 1964. Table XXXIV shows the results achieved:—

TABLE XXXIV

FLUORIDE TABLETS ISSUED IN MUNICIPALITIES IN TASMANIA FREE OF CHARGE, AND THE PERCENTAGE OF CHILDREN REACHED

Municipality	Estimated population under 16 (1964)	Number of tablets needed	Number of tablets issued in 1966	Percentage
Glenorchy	12,657	4,619,805	159,840	3.46
Kingborough	3,507	1,280,055	34,560	2.69
Devonport	5,154	1,881,210	103,680	5.5
Scottsdale	1,150	419,750	17,760	4.23
Latrobe	1,491	544,215 (136,053 for 3 months)	15,600 (3 months)	11.46
Circular Head	2,617	955,205	24,400	2.55
Burnie	5,893	2,150,945 (1,075,472 for 6 months)	143,520 (6 months)	13.34

878. However, these meagre percentages are quite illusory as indicating even a small measure of success. They of course represent tablets issued and not tablets consumed but more importantly they do not reflect the number of ' drop outs ' from each scheme, i.e., the parents or families who take an initial issue or issues but fail to persevere and run the course even for one year let alone eight or fifteen. Glenorchy has been issuing tablets for four years. Records have been kept of the families who have asked for and received issues. A reliable estimate of the turnover in families

over that period, represented by families dropping out and new ones coming in, is as high as 50% in each year, and the maximum family registration in any one year has been 656 out of an estimated 9,000.

879. Kingborough has been issuing tablets since 1964. Analysis of the records of issue shows that in 1965, 33 families obtained tablets of whom 11 only continued in 1966 and seven in 1967. In 1966, 79 families obtained tablets of whom 68 joined in that year and in 1967, 109 families of whom 55 joined for the first time. In other municipalities the scheme has been too short lived for any estimate to be made, but the initial response has always been about the 10% mark and such evidence as is available indicates not only the difficulty in awakening interest but in keeping it.

880. It did not seem, however, that tablet programmes had been publicised or pushed with any vigour. The Department of Health Services has supported fluoridation of water and has therefore refused financial support to issues of tablets, and the general municipal policy has seemed to be rather a luke-warm one of making them available without promotion. This has, I suspect, been partly due to doubts engendered by the active campaigns conducted by anti-fluoridationists as soon as fluoridation in any shape or form appears on a municipal agenda, but more importantly I think to the feeling, constantly reflected in the evidence of those with responsibilities in local government, that the matter of fluoridation was not one of water supply but of public health and outside their competence to settle. In this I think they were right. It could be that an active programme of promotion with persistent follow-up on an individual basis could improve these poor results, but experience elsewhere has been so uniformly disappointing, that I see no real prospect of any campaign, however vigorously pursued, achieving any measure of substantial and sustained success.

881. Western Australia is a State that has decided compulsorily to fluoridate. Such a decision, one would assume, would reflect an informed body of public opinion favourable to fluoridation, yet their experience in the public issue of tablets has been little better. I take Table XXXV from the submission of Mr. R. L. Taylor, Reader in Prosthodontia at the University of Western Australia. It suffers to the extent that it does not cover all the municipalities which have issued tablets in that State, as is indicated by the submission in which it appears, but no matter on what basis the nine exemplified have been chosen, the results are not without significance.

TABLE XXXV

DISTRIBUTION RATE OF FLUORIDE TABLETS BY NINE LOCAL AUTHORITIES IN WESTERN AUSTRALIA

Authority	A	B	C	D	E	F	G	H	I	Total
Children eligible	5,500	3,151	1,300	450	400	6,260	1,500	3,800	4,431	26,792
Total tablets required to provide 1 per day per month (30 days)	165,000	94,530	39,000	13,500	12,000	187,800	45,000	11,400	132,930	....
Actual average distribution per month	18,800	10,750	3,425	2,633	800	18,400	17,700	12,000	9,466	....
Maximum children for whom actual distribution would provide 1 tablet per day per month	626	358	114	88	27	613	590	400	316	3,132
Actual demand % No. eligible	11.4%	11.3%	8.8%	19.5%	6.6%	9.8%	39.3%	10.5%	7.1%	11.7%

N.B.—(1) The tablets have been distributed for varying periods—the longest 'F' for 26 months; the shortest 'D' and 'I' for 6 months.

(2) Children to be eligible for free tablet distribution must live in the area and be under 12 years of age. In 'E' the tablets are only available to pre-school children, i.e., under 6 years.

882. Such overseas programmes as have been brought to my notice have shown the same tendency. The experiment which is referred to most frequently took place in Hawaii. A three-year programme was mounted in 1957. Tablet distribution was free and the scheme and its benefits were widely publicised. In the first year response was estimated at 90%, by the third it had dropped to 12%. The conclusion expressed that '. . . fluoridation on an individual basis, although perhaps effective for a few comprehending families, is a total failure from a public health stand-point' is the only possible inference from all the available evidence[13].

883. Other extensive programmes in Germany and Switzerland have not been succesful in maintaining interest and number[14, 15]. It does not seem to be just a matter of education but rather

of the difficulty of inducing a large cross-section of the community to persist over ten or more years in a conscious daily effort. Arnold *et al.*[6] studied the response to tablets in a small select group of children, the parents of whom ' were physicians, dentists, or other professional employees of the Public Health Service who wanted their children to have the best dental care, and these children and parents were considered capable of following instructions consistently and exactly '. Results indicated the efficacy of tablet administration but ' although the persons involved in this study were, on the whole, a highly educated group, only about half of them actually continued to give their children tablets for the necessary number of years '.

884. It is not feasible to combine tablet administration with school dental programmes. The first three or four years of life are important to the success of any form of fluoridation[16] and school holidays must be taken into account. Such attempts as have been made to link tablet administration with school attendance have given results far behind those of water fluoridation[17]. As a matter of interest attempts have been made in non-fluoridated communities to benefit school children by fluoridating only the water consumed by children while at school. Because of the limited amount consumed in such cases it has been necessary to fluoridate at higher levels than normal communal levels and up to 3 ppm has been employed[18]. Some benefit has been achieved in this fashion but the disadvantageous factors already mentioned apply. Such a method of administration could also have its dangers in relation to children who as a matter of parental and independent care were receiving tablets at the same time.

885. Tablets have their place in communities without reticulated water supplies. In such places, a public health campaign to induce parents to give their children tablets in appropriate dosages from birth onwards is the best substitute for water fluoridation that is available but to ensure maximum success it will need not only promotion but vigorous follow-up action. It will give gratifying results in the case of conscientious parents, though the duration of the benefit, once tablets are stopped, is a matter of some doubt. Unless carried through into adult life, it is the prejudice to the protective effect that can result from their cessation that must always make tablets, even in the most disciplined of families, a second-best substitute.

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Section (c)—Fluoridated Salt

886. Salt has also been suggested as a suitable vehicle for the administration of fluoride. It has the advantage of being a universal item of diet, but the disadvantage that consumption is lowest at the time when the need for maximum uptake would be at its highest, namely, in the first decade of life. It has been employed on a public health basis in Switzerland apparently with some success[1] though some details of its application are still no more than experimental. Other trials have been mentioned as taking place: in Columbia and in the Netherlands, but results do not appear to be published[2]. The former is supported by the World Health Organization[3].

887. In Switzerland, reports as to its acceptance as a public health measure are incomplete and difficult to reconcile. Adler (1967)[4] citing Wespi (1962)[1] reported a 30-fold increase in the amount of fluoridated salt sold between 1955 and 1962, but Leatherman and Ellis (1963)[3] stated that the amounts bought by the public were very small and 'it was for this reason that the authorities in Zurich decided that fluoridated salt should automatically be sold unless the purchaser specially requests the non-fluoridated variety'.

888. In Switzerland, on the basis of an estimated daily average salt consumption per adult of 7 g,[4] salt is enriched by the addition of 200 mg of sodium fluoride (yielding 90 mg of fluoride ion) per kilo in addition to 10 mg of potassium iodide. It would seem that this is insufficient to achieve optimum intake, being calculated by Adler[4] to yield only 0.63 mg per adult daily and less for small children. Nevertheless this enrichment (which is nearly 50% below the desirable optimum) has led to a statistically significant fall in caries experience as indicated by the CER (DMF) tooth surfaces count after 5½ years' unsupervised use as table and cooking salt[5].

889. However, while as an alternative to water fluoridation this would seem to offer more possibility as a public health measure than anything else proposed, it cannot be said at this stage that it is a complete substitute. The doubt as to infantile benefit is a serious handicap. The incomplete results as yet to hand would indicate a caries reduction ranging from 8 to 28 per cent in the various age groups of children examined[4, 6]. These figures are significant but not so high as are to be expected from water fluoridation[7]. This may reflect the fact that at current fluoride levels the average intake would yield approximately half the optimum dose, or it may reflect other factors. For these reasons the South African Commission felt unable to recommend it[8].

890. Cremer and Büttner[9] point out that the metabolic fate of fluoridated salt may be complicated by the fact that as it is taken with meals the fluoride absorption may be reduced especially if the food is rich in calcium. They refer to a suggestion by Ericsson *et al.*[10], that sodium monofluorophosphate (Na_2PO_3F) may be a more suitable compound for use in salt fluoridation because the fluoride absorption is more rapid and less influenced by calcium ions. This is an interesting observation but I am not aware of any further work on the matter.

891. In sum the sale of fluoridated salt remains the most interesting and most feasible possibility as an alternative to water fluoridation, if one is sought. As Hardwick[11] points out:—

> 'This method of increasing fluoride intake has three notable advantages: the freedom of the individual to accept or reject this preventive measure; the slight effort it demands from the patient in following the regime; and its extreme cheapness'.

892. It has not yet been shown to be capable of achieving the full measure of protection that can be achieved with water fluoridation and there are sound theoretical reasons for assuming that it will be significantly less in younger children because of their low salt consumption. However, surveys made in relation to goitre prevention of the consumption by young children of table salt are not a guide in this instance, because salt used in the cooking and preparation of food would be just as efficient a vehicle for the administration of fluoride as free table salt. Apparently the same is not true of iodide[18].

893. There is insufficient data available on which to base any precise suggestions as to quantities, but the average calculated intake according to the Swiss figures would appear to be insufficient if it reflects the total fluoride content of the salt ingested. This would need to be watched because salt itself may have a wide and varying natural fluoride content from 0.02 to 115.1 ppm[12]. Waldbott[13] cites values for table salt of 5 ppm and for sea salt of 40 ppm. This last figure is for crude (i.e., unrefined) sea salt[14], and in this form salt has been thought to contribute a significant amount of fluoride to the diets of some Mediterranean and Eastern peoples[15, 16, 17]. However, an average daily intake of 10 g would still only yield 0.005 or 0.04 mg from this source as against the postulated intake of 1 mg from fluoridated water. On the other hand it has been calculated that in some hot Eastern countries where rock salt (200 ppm) is a prominent item of diet (20 g is the estimated average figure of daily consumption) there may be an adult intake of 4 mg a day from this source alone[17].

894. Further work would have to be done to determine the optimum amount to be added to adjust to Tasmanian levels of salt consumption and to determine the best form in which it should be added. I have no Australian figures for total salt consumption per capita. Based on salt sales and production data, they have been estimated for the U.S.A. and Great Britain at 7-10 g a day[19]. In New Zealand mean daily per capita intake as determined by survey, was 6 g in the household plus 5 g from manufactured foods, but the intake from cooking and table salt for children in an orphanage was only 2 g a day[20].

895. The New Zealand studies have shown that total salt consumption may be added to significantly by foods processed elsewhere than in the home and possibly even outside the State. I have no information as to such matters. I merely mention them as matters for technical consideration if necessary. Probably too, for infant children to achieve optimum results a programme of salt fluoridation may need supplementation by tablets.

896. While all these uncertainties remain, I feel, with the South African Commission, that it cannot be recommended, but, if water fluoridation, as the proven and most successful single measure in this field of public health, be not acceptable, then fluoridated salt would, I suggest, be the next best and a worthwhile method of achieving some success to the same end. However, unless the expedient adopted in Zurich were enforced, it would still be liable to be defeated by public indifference and ignorance. Such an expedient may be difficult successfully to put into operation and probably a better means of ensuring an adequate public consumption of fluoridated salt would be to subsidise its sale so as to make it cheaper to purchase. The small cost of fluoridating salt would still, I believe, make this a feasible proposition if water fluoridation is not proceeded with.

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Section (d)—Fluoridated Milk

897. It follows that I reject the other alternative urged, namely, that of fluoridating milk. Such a proposal, though it has been put into effect in some parts of the world[1], is fraught with difficulties and disadvantages. Either all milk is to be fluoridated or it is to be selectively done.

If selectively done it will have to be in such concentrations as will permit the ingestion of an adequate daily dose by the average child. Such information as is available as to milk consumption by children in this State indicates a highly variable intake, so variable that on the basis of individual unsupervised consumption it is difficult to suggest an optimum dosage. This variability of milk intake depending on so many factors of taste, education and economic status has been stressed as a major disadvantage in most reports[2] as well as the danger that the enthusiastic milk drinker may receive too much. The suggestion is that the variability between children is greater than it is in the case of water consumption which satisfies the basic physiological need uncomplicated by idiosyncratic or economic differences.

898. The supplemental milk issued to school children at schools in the State is one-third of a pint per child. If this is to be the chosen vehicle for administration, it would need to be fluoridated at something like 5 ppm to ensure an adequate dose for the school child drinking his ration. As milk contains 120 mg of calcium per 100 g, i.e., 1,200 ppm, as well as other ions and solids, some metabolic loss due to precipitation or complexing is likely[3, 4, 5] though its extent is uncertain. However, it has been established that fluoridated milk will lead to an increase in available fluoride[6, 3, 7, 8] though quantitative data are meagre and the measure of its long-term efficacy is still uncertain[8, 9].

899. If milk in the home is to be used as the vehicle of administration because of the inadequacy of the school milk issue in relation to pre-school children and to cover weekends and holidays, then it has no advantages over and all the disadvantages of tablets. It would, moreover, be more costly for the parents.

900. But the administrative difficulties would in my opinion be insuperable. Fluoridation would have to be carried out in milk processing plants and they presumably would have to be responsible for the analytical determination of fluoride content. This with milk is a tedious and difficult business as the relatively straightforward colormetric methods used for the primary analysis of water are not suitable for milk which requires evaporation, ashing, distillation and titration[10, 9]. Moreover it would, I am informed, take approximately 24 hours to complete the analysis of each sample.

901. I concur without reservation in the conclusion of the South African Commission that the disadvantages of milk as a vehicle for fluoridation in a public health programme make it ' one of the least suitable methods available for administering fluoride '[9].

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Section (e)—Miscellaneous, Topical Applications, Dentifrices and Dental Manpower

902. I am not by my terms of reference called upon to comment upon the efficacy of other fluoride weapons in the dental armoury, though I have heard much evidence as to their effect. Strictly speaking matters such as painting the teeth with fluoride solutions (topical application), the use of fluoridated toothpastes and powders and fluoride mouthwashes are useful supplements, but in no sense a substitute for fluoridation. Their effect is post-eruptive and topical only, and

unless swallowed, which they should not be as the fluoride concentration may be many thousand times that of fluoridated water, they do nothing for the tooth in its critical stages of pre-eruptive formation and calcification. Moreover, topical application is a clinical procedure to be administered only by skilled personnel. It has to be repeated at intervals of six to twelve months and is therefore both costly and demanding of professional time. All have been proved efficacious in some degree under controlled conditions[1, 2, 3, 4, 5], but no one has suggested that they constitute an alternative public health measure to fluoridation.

903. I am not called upon to report on the adequacy of the dental manpower available in this State to cope with the carious tide either now or in the future, though such matters necessarily intruded into the evidence. I merely state that the ratio of dentists to population is obviously and substantially below that accepted as desirable in dentally enlightened countries, and the evidence indicates that it will worsen and not improve. The provision of a dental nurse scheme based on New Zealand lines will not overcome the problem (despite forty years operation it has not done so in that country) though it is to be commended as alleviating the current sorry plight of the younger age groups of the community. I understand that the matter of the need for a Dental School in this State is to be or is being considered by a Departmental Committee. I heard a great deal of evidence on the matter, not unnecessarily, because the dental manpower position was relevant to the case for fluoridation, and I record for information that my impression of that evidence was that it presented a very strong case indeed for the establishment of such a school at the earliest opportunity if the position in this State in relation to dental services, both for the child and the adult, is not further to deteriorate.

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(vi) JACKSON, D. and SUTCLIFFE, P.—'Clinical Testing of a Stannous Fluoride-Calcium Pyrophosphate Dentifrice in Yorkshire School Children'.

[3] STOOKEY, G. K.—'Fluoride in Drugs Ingested as Anti-cariogenic Agents'. (1967). Unpublished Contribution to provisional text of a Monograph on 'Fluorides and Human Health' in course of preparation by World Health Organisation.

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[5] Republic of South Africa.—'Fluoridation'. Report of Commission of Inquiry, §§ 173-184. Govt Printer, Pretoria. 1966.

PART M—TERMS OF REFERENCE—QUESTION 5

'What would be the cost of adding fluoride to water supplied to the public and whether as a public charge it would be justified in the public interest.'

CHAPTER 53. COSTS

Section (a)—General Considerations Affecting Costs

904. The cost of fluoridation will vary from region to region, municipality to municipality, the variables being mainly the number of installations required, whether they can be combined with other plant required for water treatment, the type of chemical used and local problems of operation and maintenance.

905. None of these are matters of difficulty but they raise individual problems of engineering design which may produce quite wide variations in cost. The figures which follow therefore are guides only, the actual costs can only be satisfactorily estimated on a particular basis. I did not feel justified in asking for such estimates to be specially prepared for the purposes of the Commission, particularly in view of the uncertainty that I feel attaches to the future pattern of communal water supply in this State. But I have taken advantage of and reproduce for information, such surveys as have been made.

906. In talking of uncertainty I have in mind that the present pattern of water supply in this State would appear to be transitional, in that the trend facilitated by the Water Act 1957 and the Metropolitan Water Act of 1961 towards the aggregation of water supply authorities into Regional or Metropolitan schemes may well continue so as to embrace in larger schemes some municipalities which at present operate as independent authorities. Apart from the possible outcome of any recommendations of the Municipal Commission under section 14 of the Local Government Act 1962, this process is, I understand from the evidence, likely to be accelerated by a growing recognition of a need for water treatment (apart from fluoridation) to get rid of pollution and other contaminants affecting the quality of municipal water supplies. There are also in some cases substantial questions as to the adequacy of small local schemes to serve increasing populations yet to be resolved.

907. These problems exist, I am told, and they are increasing. In the past they have in some municipalities been ignored, or at least no really sophisticated efforts have been made to control them.

908. Where a municipality is served by small and multiple sources of supply, the cost of water treatment can be disproportionate; in fact the recognition of this was acknowledged in evidence as the reason why stricter measures have not been taken to enforce improvement on some existing supplies. Considerable economies, in installation, operation and maintenance, can be gained if a single large plant can take the place of multiple smaller plants, and if all aspects of water treatment can be included in one comprehensive scheme. Thus if chlorination is to be introduced to get rid of pollution, fluoridation will be cheaper than if introduced without it. It is also quite clear that considerable economies in fluoridation costs could be effected in some parts of Tasmania if small municipal water supplies were to be aggregated into larger regional schemes. Statutory powers which could be used to this end already exist in Section 16 of the Water Act of 1957. In fact a comprehensive survey[1] made by the Rivers and Water Supply Commission for the purposes of this Commission as to the practicability of fluoridating all municipal water supplies of the State has made it clear that in the case of a few municipalities with multiple, scattered, supply sources, the cost of fluoridation might be considered excessive in the absence of some regional scheme or sharing of operating staff where proximity made this possible. Examples are Circular Head, Huon and Ringarooma. Such plainly excessive costs obtain however only in a small minority of the schemes examined, though economies should always follow where it is possible to aggregate small sources of supply.

909. I am mindful of the fact that no general questions relating to the water supplies of the State have been committed to my charge; I give my own appreciation of the position solely to explain the rather general way in which I have approached this particular aspect of my commission.

910. To deal briefly with the other variables on which costs will depend—

(*a*) The output of the plant, which will of course depend on the population served, will generally determine the type of chemical used. Sodium fluoride with its high solubility seems generally to be preferred for the smaller plants, whereas the

cheaper sodium silico fluoride with lower solubility is preferred for the larger[2]. The relative costs (1967 figures) are $294 and $160 a ton respectively and it takes 22.5 lbs of sodium fluoride or 16.7 lbs of sodium silico fluoride to fluoridate one million Imperial gallons to the level of 1 ppm. Calcium fluoride is of course much cheaper than either, but solubility is very low and consequent engineering difficulties are considerable and, though it has been employed in artificial fluoridation, it is not recommended on that account.

(*b*) There are at present no local supplies of fluorides to be drawn upon and the present Tasmanian sources are Scandinavian. These costs could be varied if local or Australian supplies became available; thus in N.S.W., hydrofluoric acid is being employed in the fluoridation of Sydney's water, the choice being due, I am informed, to the savings effected by using a local source of supply.

(*c*) The method of mixing is generally determined by the scale of the plant, i.e. the solution feed is preferred for small installations while volumetric (dry) feed is preferred in larger installations. Neither present any technical difficulties and there is a large range of equipment available for the operation of either system. In general the solution feeders may be simpler and cheaper, but in larger plants this would be offset by the size of the solution tanks required and other factors. Volumetric (dry) feeders are fairly sophisticated types of machine and experience in Hobart would indicate that their periodical inspection, maintenance and repair is a matter for a skilled electronics technician. In Hobart the amount of time involved would be two to four hours every couple of months for periodical checks plus whatever time may be necessary for actual repairs and adjustments. The services required in this regard would be a matter of independent contract and not of employment and are not a large factor in the operating costs. They do point however to the advantages of technical supervision by a central authority. Solution feeders in smaller plants are relatively simple and do not require the same level of technical assistance to be available when required.

(*d*) Operating costs, excluding materials, are mainly those of the provision of staff to supervise the functioning of the machines (these are automatic with fail safe devices in the case of breakdown so that supervision may be intermittent), to handle the raw material, to conduct the daily analyses as a check against misfunction, and to collect samples for periodical analysis by the Government Analyst. The daily analysis is colorometric, i.e. it is performed by matching a sample to which a reagent dye has been added with a solution, similarly coloured, of known strength and though a matter of responsibility it is not a lengthy process and is a matter of care rather than knowledge. Other more sophisticated methods of analysis are the responsibility of the Government Analyst and would not be conducted at the plant. Subject to the periodic supervision of the Government Analyst, the operation of a fluoridation plant does not call for formal training in chemistry, or skills exceeding in degree those found in other skilled trades. The cost is therefore dependent upon whether factors such as the number of inlet points and the distances between them, make it necessary to employ a full-time operator as in Hobart with its three separate plants, whether the operator can perform other duties or be shared with another municipality or whether, as in Launceston, with its one plant additional staff can be dispensed with and it can be quite satisfactorily handled with unskilled assistance from the laboratory of the water chemist. The economic advantage is clearly on the side of the larger single supply. As an instance, apart from the chemical used, I am informed, it would cost little more to fluoridate all the cities and municipalities drawing water from the West Derwent and Southern Regional Schemes than it does at present to fluoridate Hobart only[3].

911. These are general considerations. Of all the factors mentioned the cost of the raw material is undoubtedly the least important. The combined daily output of the three reservoirs supplying Hobart for the years 1964-65 and 1965-66 averaged approximately 7,000,000 gallons a day. To fluoridate that at 1 ppm would take approximately 117 lbs of sodium silico fluoride which at $160 a ton would cost a little more than $8.

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1 Rivers and Water Supply Commission. Collation of Particulars Submitted by Municipalities, throughout the State, for information of the Royal Commission on Fluoridation. 1967.

2 LONGWELL, J.—'Chemical and Technical Aspects'. *Roy Soc. Hlth J.*, 77: 361: 374. 1957.

3 Director-General of Health Services, Tasmania. Report of the Director-General of Health Services for the year ended 30 June 1964 at p. 35. Govt Printer, Hobart. 1964.

Section (b)—Costs of Tasmanian Schemes, Present and Projected

912. I have obtained evidence of the costs of schemes in operation in Tasmania. They are as shown in Table XXXVI.

913. I have been given estimates of the cost of fluoridating other Tasmanian water supplies. Such estimates would by comparison with known costs of existing schemes in Tasmania and with known and estimated costs for schemes in other parts of Australia and elsewhere appear to be high. They are, however, based on water schemes as they existed at the time the estimates were made. They assume fluoridation as an individual municipal exercise, self-contained in relation to staff and equipment, and do not reflect the economies that could be obtained by the aggregation of supplies in regional schemes or the provision of fluoridation not as an isolated operation but as part of a comprehensive scheme of water treatment. They lead inevitably to the suggestion that if fluoridation is decided upon, there are substantial advantages economic and otherwise to be gained by putting it in the hands of a central water authority which will be able with technical competence to integrate fluoridation with all other aspects of water treatment, with plans for regional supply and shared technical staff. For instance, it is clear that if fluoridation for the City of Glenorchy and the Municipality of Clarence were desired, fluoridation at source (i.e. at Bryn Estyn and Lake Fenton) by the Rivers and Water Supply Commission and its offshoot the Metropolitan Water Board (which now controls the supplies of approximately one-third of the State's population) would possess technical and economic advantages not only for the City and Municipality mentioned but for Hobart as well.

TABLE XXXVI

COSTS OF OPERATION OF FLUORIDATED WATER SUPPLIES IN TASMANIA

City or Area and population served	*Capital Costs incl. Plant and Buildings* $	*Annual Operational Cost*	1965/66 $	1966/67 $	*Amount payable per annum by City after Treasury Subsidy*	*Cost per head of population of city per annum after subsidy*	*Remarks*
Hobart (53,226)* may include 1,000 to 1,500 in Mt Nelson and Fern Tree areas not receiving fluoridated water (T. 1775)	30,258 (1964)	Interest and sinking fund	3,439	3,439	1966/67		Three dry feeders at reservoirs. Fluoridation at source not possible because unfluoridated Municipalities served from same sources. This makes it dearer. Full time operator employed. Operation and Maintenance costs increased because of multiple dispersed fluoridation points.
		Chemical Na_2SiF_6	4,600[2]	3,120	$		
		Power, Maintenance	900	784			
		Operation, wages, analyses, transport and sundries	4,941	5,123	5,610[4]	10.6c	
			13,880	12,466			
Launceston (37,210)*	19,716 (1962)	Interest and sinking fund	2,172[1]	2,172[1]			One dry feeder at water works only. Operation, analyses etc. carried out by water operating staff, under the supervision of waterworks chemist who supervises complete water treatment plant. No special operator for fluorides employed.
		Chemical Na_2SiF_6	2,059	2,039			
		Power, Maintenance	529	519			
		Operation, wages, analyses, transport and sundries.	932	1,068			
			5,692	5,798	3,985[3]	10.8c	

City or Area and population served	*Capital Costs incl. Plant and Buildings* $	*Annual Operational Cost*	1964 $	1965 $	*Amount payable per annum by Municipality after Treasury Subsidy*	*Cost per head of population of municipality per annum after subsidy*	*Remarks*
West Tamar (Beaconsfield and Riverside Beauty Point since 1962). (9,972)* (9,000)†	2,318 (1962)	Interest and sinking fund	250[1]	250[1]	$		One dry feeder operated at complete water treatment plant. Operation, analyses, etc. carried out by water operating staff under supervision of water works chemist who supervises plant.
		Chemical Na_2SiF_6	258	250			
		Power, Maintenance	25	38			
		Operation, wages, analyses, transport and sundries.	307	422			
			840	960	605[3]	6.7c	
			1962 $				
Beaconsfield (Discontinued 1962) (2,500)†	330 (1953)	Chemical Na_2SiF_6	73				One wet feeder operation analyses etc., carried out by water works chemist who operated water treatment plant.
		Power and Maintenance	19				
		Operation, wages, analyses, transport and sundries	107				
			199		100[3]	4c	

* Census 1966. † Population estimated to have been served.

[1] Supplied by Treasury.

[2] Includes surplus chemical purchased.

[3] State subsidy under Ministerial authority dated 9 January 1959, amounts to 50% of annual costs, the capital cost having been met by the Treasury. This authority is not applicable to any new schemes.

[4] State subsidy at the moment under Ministerial authority dated 12 September 1962, amounts to 55% of annual costs only.

TABLE XXXVII

ESTIMATED COSTS OF OPERATION (1967) OF FLUORIDATION PLANTS IN SOME TASMANIAN WATER DISTRICTS

District and Population	*Capital Costs including plant and buildings*	*Cost per Head of capital cost*	*Annual Operational Costs*		*Amount payable per annum by Municipality after Treasury subsidy*	*Cost per head of population per annum after subsidy*
	$	$		$	$	
Latrobe (3,000)	6,200	2.07	Interest and sinking fund	496		
			Chemical Na_2SiF_6	100		
			Power, Maintenance	130		
			Operation, wages, analyses, transport and sundries	1,000		
				1,726	777	25.9c
Geeveston (2,100	9,000	4.29	Interest and sinking fund	720		
			Chemical Na_2SiF_6	240		
			Power, Maintenance	220		
			Operation, wages, analyses, transport and sundries	1,400		
				2,580	1,161	55.3c
Dover (600)	3,500	5.83	Interest and sinking fund	280		
			Chemical Na_2SiF_6	100		
			Power, Maintenance	125		
			Operation, wages, analyses, transport and sundries	1,200		
				1,705	763	$1.27
Queenstown (5,100)	9,600	1.90	Interest and sinking fund	768		
			Chemical Na_2SiF_6	300		
			Power, Maintenance	200		
			Operation, wages, analyses, transport and sundries	1,000		
				2,268	1,022	20.1c
Bothwell (600)	4,000	6.66	Interest and sinking fund	320		
			Chemical Na_2SiF_6	50		
			Power, Maintenance	90		
			Operation, wages, analyses, transport and sundries	900		
				1,360	612	$1.02
Cygnet (1,500)	8,000	5.33	Interest and sinking fund	640		
			Chemical Na_2SiF_6	100		
			Power, Maintenance	125		
			Operation, wages, analyses, transport and sundries	1,000		
				1,865	8.39	56c
Scottsdale (3,000)	5,000	1.66	Interest and sinking fund	425		
			Chemical Na_2SiF_6	480		
			Power, Maintenance	200		
			Operation, wages, analyses, transport and sundries	900		
				2,005	9.02	30c (1963)

The interest and sinking fund is assessed at 8% per annum of capital costs. The subsidy at the moment under Ministerial authority dated 12 September 1962 amounts to 55% of annual costs.

914. I set out in Table XXXVII the estimates to which I refer. Because I feel strongly that any decision to implement fluoridation should be undertaken under central direction and supervision, I think they are not only hypothetical but rather unreal. I think they should only be regarded as related to actual costs if an immediate decision were made to fluoridate in the municipalities named on a wholly municipal basis irrespective of future patterns of water supply and

of the economies and other advantages that could be obtained under central direction. However, such advantages might not be gained if there was a regionalisation of supply, but the decision whether or not to fluoridate was left to be taken independently at a lower level by the municipalities concerned. I am satisfied these are not speculative considerations. I have heard a great deal of evidence as to the present inadequacies of water systems in the State and technical considerations which may guide their future development. I am not in a position to anticipate the effect that political considerations may have upon such documented proposals as exist. Nevertheless the evidence is cogent enough to make me doubt the wisdom as a practical exercise of forecasting costs based on present patterns of supply.

915. With these considerable reservations I show in Table XXXVII estimates as at 1967 of the costs of fluoridating a number of Tasmanian municipalities. I have incorporated in the Table a consultant engineer's appreciation of the engineering factors involved, but have revised his cost figures so as to accord with the present statutory position as to subsidy (sec. 61 (5), The Public Health Act 1962).

916. As an instance of the economies to which I have previously referred, the consultant engineer responsible for these estimates, revised the figures for Latrobe in the light of a proposed regional scheme which would take in that municipality. The estimated saving was not less than 50%[1]. As Latrobe was one of the easiest and simplest to fluoridate on a municipal basis even higher savings might be anticipated in other municipalities where more complex supply problems exist. Another estimate, pointing directly to the advantage of regional schemes, was that fluoridation at the headwaters of the North Esk Regional scheme would save up to 80% of both capital and operating costs estimated as likely to be incurred if municipalities served by this scheme fluoridated separately.

917. I have stressed the uncertainties of this rather hypothetical exercise of assessing probable costs. In summary subject to the qualifications mentioned and guided by the evidence of the consultant engineer referred to, I would think the likely cost of fluoridating Tasmanian municipalities not at present so served, on the basis of their present supplies to be as shown hereunder. Because of the wide range of factors, other than population, which may affect supply, I do not feel justified in attempting to make my figures any more precise:—

ESTIMATES OF COST PER CAPITA PER ANNUM OF FLUORIDATING TASMANIAN MUNICIPAL WATER SUPPLIES

	With Population over 3,000	*With Population* 1,000-3,000	*With Population* 200-1,000
Total cost per capita	45-90 cents	90 cents to $1.30	$1.10 to $4.40
If reduced by Government subsidy of 55%	20 to 40 cents	40 to 60 cents	50 cents to $2.00

918. The evidence is that it would not generally be economic to fluoridate supplies serving less than 200 consumers and as the table suggests there will be cases above that figure where the same result may follow. Always it is the labour involved in daily testing and supervision which is by far the most expensive item. In America it has been found possible in the case of small communities (population from 70-1,000) by utilising a simple design of solution feeder, centrally serviced, successfully to reduce annual per capita costs to $1.22[2], so these Tasmanian figures should not be regarded as absolute. Capital costs were saved by installing simple constant rate feeders and test analyses were limited to periodical instead of daily checks. However, it is felt that the most satisfactory way of reducing costs for small Tasmanian communities is by the extension of fluoridation on a regional basis.

919. By way of contrast to these estimated Tasmanian figures I refer to published and other figures for other communities expressed in the currency of the country concerned:—

(*a*) U.S.A. large city supplies—average cost (1959) 7.9 cents per person per year (range 12.6 cents to 1.8 cents)[3, 4].
(*b*) England (1962)—10d per head per annum[5].
(*c*) Netherlands (1963)—5-8c (40 cents during the initial period[6].
(*d*) New Zealand (1957)—1s per capita[7].
(*e*) South Africa (1967)—10.15 cents per person per year[6].
(*f*) Canada—10 cents per capital per annum[8].
(*g*) Other Australian States—as in Table XXXVIII.

TABLE XXXVIII
COMMUNITIES SERVED BY FLUORIDATION—AUSTRALIA

Community	*Population Served*	*Year Installed*	*Costs $ Australian*			
			Installation		*Operation*	
			Cost per Head	*Total cost*	*Cost per Head*	*Total Cost*
			$	$	$	$
New South Wales—						
Bega	3,920	1965	1.69	6,640	....	....
Condobolin	3,340	1963	1.00	3,200	0.19	600
Cooma	9,350	1963	0.84	7,600	0.21	1,882
Forbes	7,150	1965	0.50	3,576	....	....
Goulburn	20,180	1961	0.32	6,764	0.07	1,500
Grafton	40,000	1964	0.22	8,680	0.10	4,000
Griffith	17,190	1965	0.74	12,690	....	....
Hay	4,170	1965	1.48	6,188	....	....
Nambucca (Shire)	9,040	1964	0.30	2,400	0.09	750
Nyngan	2,650	1966	1.04	2,758	....	....
Orange	20,000	1962	0.58	11,644	0.17	3,300
Parkes	8,610	1965	0.86	7,438	....	....
Queanbeyan	11,670	1964	....	....	....	....
Tamworth	21,180	1963	0.55	11,000	0.22	4,500
Yass	4,000	1956	0.13	500	0.12	460
Sydney (estimated)	3,000,000	1967	0.14	378,500	0.06	170,000
Victoria—						
Bacchus Marsh	3,500	1962	0.46	1,600	0.04	500

Table produced by Dr M. J. Flynn[9].

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[7] New Zealand.—'Fluoridation of Public Water Supplies'. Report of Commission of Inquiry, §§ 436-437. Govt Printer, Wellington, New Zealand. 1957.
[8] Ontario.—Report of the Committee appointed to inquire into and Report upon the Fluoridation of Municipal Water Supplies, p. 94, § 197. Ontario Water Resources Commission. 1961.
[9] FLYNN, M. J. DR..—Table. 'Communities Served by Fluoridation'. Produced in evidence. Transcript p. 1432.

Section (c)—Whether Such Costs would be Justified in the Public Interest

920. I am further directed to inquire whether the cost of fluoridation as a public charge would be justified in the public interest. There are two difficulties in the way of a straightforward answer, the first the absolute one of measuring in monetary terms an improvement in public health, the second, and more relative, whether cheaper means can be found to the same end.

921. The first is a matter of equating dissimilar values. So far as this can be done, I believe it is amply justified. The average cost of the three existing Tasmanian schemes is 17 cents per

head of population served per annum. If for future schemes this went as high as $1 per head, it would still be less than the cost of one filling per head per year without taking into account the intangible benefit gained by lessening, over the community as a whole, caries-caused pain, inconvenience and debility.

922. More concrete figures are available from New Zealand. Fluoridation was introduced in Hastings in 1954. In 1966 dental officers of the New Zealand Department of Health made a survey of the effectiveness of fluoridation by assessing—

(1) The difference in the cost of treatments by the School Dental Service in Hastings as compared with other unfluoridated centres.

(2) The difference in the number of fillings needed by each child to complete all fillings and extractions noted as required at each six-monthly inspection (including both deciduous and permanent teeth).

(3) The change in the number of children whom dental nurses were capable of treating before and after fluoridation.

The survey[1] showed:

'(1) The cost of dental treatment, i.e. for fillings and extractions per " Completion " in Hastings children has been reduced from 8.57 dollars to 4.27 dollars. Total treatment costs have been reduced from 11.45 dollars to 7.48 dollars. The latter figure includes a charge for examination and X-rays, this charge being a standard one that is independent of the treatment needs of the individual.

'(2) The number of fillings required in the deciduous teeth of Hastings children has been reduced from 5 to 3 per completion; and the number of fillings required in 12-year-olds (who were selected as an example) has been reduced from 5 to 1.2 fillings per completion.

'(3) The usual number of children whom Hastings nurses were able to treat prior to fluoridation was about 450 per nurse. This number has been increased to 700 children per nurse and in the case of experienced operators 800 to 900 children are being handled.'[2, 1].

923. I believe these figures to be a satisfactory indication of the benefits that might be expected under Tasmanian conditions. In fact they might be conservative. The average cost of restoring a tooth (once) in this community is estimated at $4 and extractions at a similar figure. Molars have up to five surfaces all of which may at one time or another need restoration. Artificial restorations, dentures, crowns, etc., cost much more. If dental treatment for children alone was reduced by fluoridation by only 40%, it is obvious that there should be a considerable community saving.

924. I have elsewhere (Part ' L ' Ch. (52)) expressed the opinion that alternatives to water fluoridation such as fluoride tablets or fluoridated milk are not and could not be expected to be efficacious as a public health measure. It therefore becomes unreal to attempt in their case to measure cost against cost, as the comparison would only be useful if reasonably comparable results could be expected. I have the further difficulty that, due no doubt to the meagre results obtained, no attempt at analysis of the administrative costs involved in such schemes as have been attempted in this State has been kept.

925. However, some idea of the cost of a tablet programme could be obtained from the following figures. Fluoride tablets wholesale (1966) cost anything from 12 cents to 50 cents a hundred[3]. I have evidence that in 1965 Councils and Government Departments could obtain bulk supplies at 2s per bottle of 120 (retail price 4s 6d). Allowing each child one tablet a day gives a starting figure of 6s or 60 cents as the cost of tablets per child per annum. Children under twelve comprise 28% of the population[4]. Applied to Hobart (population 53,200) this gives an approximate figure of 15,000 children per year that would need tablets at 60 cents per head which comes to $980, say $1,000 in round figures. This allows nothing for the administrative costs concerned with distribution, recording and follow up. In the absence of an efficient and rigorous programme embracing these three matters, experience shows plainly that the programme would be largely futile. I have no Tasmanian figures which help me here. I have some evidence that Western Australian experience indicates that the administration costs involved in distribution are at least equal to the costs of the tablets but this allows nothing for follow up. However at $2,000 there is plainly a big nominal saving against the fluoridation of water supplies and the saving will still

be substantial even if tablets were continued up to the age of fifteen to cover the period of post eruptive maturation. But the paper saving thus demonstrated will be lost and a substantial debit incurred the other way, if, at the public charge, the tablet programme be supplemented, as I believe it should be, by topical applications, administered clinically every six months for each child over the age of six years (Ch. 52, sec. (b), *supra*).

926. But the real argument against tablets is not economic. It is simply that as a public health measure they do not work (see Ch. 52, sec. (b), *supra*), that is to say, while tablets may be effective as a caries prophylactic for the individual if continuously and unfailingly taken every day for at least the first fifteen years of life, their distribution as a public health measure presupposes a standard of parental care and childish discipline which no tablet programme has yet been able to achieve. Those who will succeed are those who would probably take dietary and hygienic measures anyway to protect their children's teeth; those who will inevitably fail will be those who are most in need of the assistance not depending on conscious effort that fluoride gives to their dental health. Further such a programme excludes from consideration, any adolescent or adult benefit to be gained from the continuous ingestion throughout life of fluoridated water and it ignores the benefits postulated for the aged in the reduction of osteoporosis and possibly aortic calcification though these may not be regarded as decisive factors.

927. In my considered opinion no true balance can be struck between the efficacy of water fluoridation as a public health measure and the cost of a fluoride tablet programme.

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[4] Census of the Commonwealth of Australia. Census Bulletin No. 61: 1: Summary of Population—Tasmania. Commonwealth Bureau of Census and Statistics, Canberra. June 30, 1966

PART N—TERMS OF REFERENCE—QUESTION 6

'Whether the existing Statute Law of this State and in particular the statutory scheme embodied in Sec. 61 of the Public Health Act 1962 is adequate in scope and effect to allow of the implementation of any decision to add fluoride to water supplied to the public and if not, what amendments are necessary and desirable.'

CHAPTER 54. WHAT STATUTORY AMENDMENTS ARE REQUIRED

928. The key to this question is the phrase 'any decision to add fluoride'. I have already expressed the view (Ch. 51, *supra*) in relation to the third question asked by my Commission that the decision whether or not fluoridation should be undertaken in this State as a mandatory public health measure is one for Parliament. If Parliament's decision should be permissive, merely leaving it to the discretion and courage of local authorities, which is roughly the present position, then no major changes in present legislation are needed. Fluoridation will continue to be, as it is now, a matter of local politics and the functions of the present Fluoride Committee will continue to be exhortatory and advisory. In such circumstances and in the light of its workings, there would appear to be little point in attempting to remould it, indeed I question that it serves any purpose at all that could not just as usefully be served by officers of the Public Health Department and the Rivers and Water Supply Commission, if necessary as an Inter-Departmental Committee, without the trappings of a statutory body lacking any real executive power. If, on the other hand, Parliament should decide that fluoridation be undertaken as a matter of direction as soon as it is feasible to do so in any particular case, then the present legislation is clearly inadequate. Until this broad question of policy is decided, consideration of future legislation is perhaps premature, though the following matters could become relevant.

929. If the decision should be that the water supplies of the State shall, in the interests of public health be fluoridated where and as practicable, then I see the implementation of such a decision being entrusted to two authorities:—

(*a*) It should be the responsibility of a Fluoride Committee to decide when, where and to what extent fluoridation should be put into effect, to supervise the dental and health aspects of the operation and to keep the practice of fluoridation under surveillance.

(*b*) It should be the responsibility of the Rivers and Water Supply Commission to give effect to the decision, utilising statutory procedures and powers amended only so far as may be necessary to make it clear that fluoridation of drinking water is within the powers and functions conferred on the Commission by Secs. 16, 17 and 110 of the Water Act 1957.

930. To serve the purposes envisaged by the preceding paragraph the Fluoride Committee should be reconstituted. It should operate within the Department of Public Health which should provide all necessary administrative and clerical assistance. As its functions are technical, expert and advisory I see no need for it to comprise any more than the following—

The Director of Public Health who shall be Chairman and whose Department shall supply such administrative clerical and other assistance as shall be necessary.
An Engineering Member of the Rivers and Water Supply Commission.
A Dental Member.
A Biochemist.
A Consultant Physician who may be a Professor of Medicine.

931. I have omitted the Government Analyist. I am not satisfied that the present arrangement whereby the Analyst conducts the check analyses and reports to himself, as it were, as a member of the Fluoride Committee has worked well. There would appear to have been a failure on the part of the Committee generally to realise just how far fluoridation programmes were falling short of planned optimum levels (an explanation given by one member was that their role was permissive but did not entail supervision), nor was I satisfied with the methods used for the routine collection and bulking of samples for testing. If test analyses are necessary—and public relations if nothing else suggest that they are—the collection of samples should as a

matter of scientific discipline be randomised, not always taken at the same time from the same tap and they should be separately preserved so as to admit of the identification of the source of difficult or doubtful specimens. Procedures adopted in the past have not achieved these aims. Further, I consider it advisable that the Analyst should maintain his status as an independent authority (you do not appoint an auditor to the Board which he is supposed to audit) and the Committee in its turn should bring a critical and independent mind to the matters which his analyses are supposed to take care of.

932. It is easy to suggest other additions to the Committee but I would reject them unless it can be seen that the functioning of the Committee will be impeded without them. What is required is a small technically competent advisory committee, expert in the subject, and skilled in the interpretation and understanding of the matters referred to them. They would exist to advise the Minister when and how it was feasible to implement a policy decision already made by Parliament and to exercise, as his advisor, public health surveillance over the results.

933. There is no need therefore for a larger unwieldy body, representative of a greater number of interests, which might be required were their functions more deliberative. I have given some thought to three specific matters—

(*a*) While the Committee will need to keep in touch with the clinical impressions of the practising medical and dental profession I do not think this is necessarily achieved by adding a busy practising member, whose practise and interests may have little to do with the subject, nominated as a representative of some professional body. I consider there is ample provision to this end in the recommended appointment of a dentist and a consultant physician. The advice of the latter would also be invaluable in the sifting and testing of complaints of untoward effects.

(*b*) The last Committee had a local government representative of the Municipal Association. In ordinary circumstances he could contribute nothing of value to the Committee and I do not recommend such an appointment.

(*c*) There are other Government Departments concerned with the problem of fluorides in industry, viz., the Department of Labour and Industry and the Mines Department. Experience at George Town, I suggest, has shown a particular need for consultation with such Departments, and, though this is outside the scope of my Report, I do recommend that consultation should be undertaken even if this Committee never comes into being. Under Ministerial direction, suitable arrangements for co-operation of all Departments concerned should be possible without adding Departmental representatives as members (see Ch. 27, *supra*).

934. The functions of the Fluoride Committee should be:—

(*a*) To advise the Minister whether or not it is practicable to fluoridate any given water supply; the method to be adopted and the chemical and concentration to be used and, if it is not recommended because of engineering or economic difficulties, what alternatives, if any, should be adopted and how they should be brought into effect.

(*b*) As a preliminary to such advice it should conduct in every case of a projected fluoridation scheme a feasibility study surveying—

(i) On the basis of information available to the School Dental Service and employing sampling procedures if necessary, the state of the dental health of the community to be served with particular reference to mottling.

(ii) The advice of the Rivers and Water Supply Commission on all water engineering aspects relative to the project under consideration, including the feasibility of combining fluoride with other aspects of water treatment and any considerations arising from possible or projected schemes for regionalisation of water supplies. Such matters are finally the responsibility of the Rivers and Water Supply Commission but they are relevant to any decision to fluoridate.

(iii) The question whether there exists in respect of the municipality or region to be served any industry in which or as a result of whose operations fluorides are ingested, produced or emitted in quantities sufficient to raise the possibility that with water fluoridation they may constitute a hazard to health and what steps have been taken to ascertain its extent and to overcome its effects.

(iv) Any medical or other special problems that to the knowledge of the Committee are relevant.

(v) Such other matters as it may have been directed to consider by the Minister.

(*c*) If present policy of subsidising municipal expenditure on fluoridation, be continued, the Committee should then recommend the amount. I have in mind that there should be variable rates, favouring smaller municipalities on whom the financial burden may be a lot heavier. The considerations underlying my recommendation in this regard appear in the preceding chapter. It could be however that considerations previously discussed (Ch. 51, *supra*) will be seen as justifying an assumption of the full cost of fluoridation by central authority.

(*d*) To keep fluoridation schemes when introduced under surveillance and for that purpose—

(i) To meet at least once every six months unless the holding of such a meeting is dispensed with by the Minister. It could be that doubts as to its functions were the cause of the previous Committee not meeting at all for over two-and-a-half years, but such an interregnum would be quite incompatible with the role of surveillance that I recommend it should exercise for the future if fluoridation be accepted.

(ii) To review periodically but at intervals of not less than every six months, the level of fluoridation maintained as disclosed by analysis both at the waterworks and by the Government Analyst.

(iii) To inquire into and take such action as may be necessary to adjust discrepancies disclosed by any such reports.

(iv) To receive and inquire into complaints and to advise the Minister thereon.

(v) Periodically to require a report from the School Dental Service as to the incidence of dental fluorosis in a fluoridated district. Such a report might be based on clinical reports but at intervals of not less than three years it should be confirmed by an examination of a sample population of children of such size and age as may be determined by the Committee. In the case of any report pointing to a prevalence of dental fluorosis which may be thought to exceed a level represented by a community index of .04 fixed by reference to the methods of Dean to decide what further steps should be taken to determine its extent, its causes and the measures necessary for its abatment.

(vi) Periodically but at intervals of not less than three years to require surveys to be conducted as the responsibility of the School Dental Service adequate in size and scope to determine the effects of fluoridation on the dental health of school children, and in the light of such surveys to determine whether any adjustment is needed in the level of fluoridation.

(*e*) To inform itself of the fluoride content of fluorinated pharmaceuticals and drugs and to make such recommendations as it sees fit to the Minister in relation to their sale, prescription, packaging and use.

(*f*) Generally to inquire into and advise upon any change in industrial conditions, industrial processes, foodstuffs, dietary habits and any other matters, whether related to the foregoing or not, which could have the effect of augmenting fluoride intake in a fluoridated community, either by individuals or by the community as a whole.

(*g*) Formally to report on all matters coming under its notice or committed to its charge once yearly but oftener in relation to specific matters if so required by the Minister.

935. On receipt of the advice of the Fluoride Committee, tendered as the result of a feasibility study along the lines indicated above, then, if Parliament has so determined, it should be the responsibility of the Minister, to issue a direction to the Rivers and Water Supply Commission to put the decision into effect. In doing so, the Rivers and Water Supply Commission should act as it does or may do now either by its own officers or by arrangement with other Departments or Municipalities (Sec. 22). I prefer that there should be one executive authority only. Other water authorities such as the Metropolitan Water Board should be subject to the same powers of direction, etc., by the Commission as are Municipalities under Sec. 22.

936. It follows that all matters relating to the provision and training of water operators should remain the responsibility of the Rivers and Water Supply Commission and not the Fluoride Committee. In this way the integration of all water treatment processes should be facilitated. It would seem that beyond a small amendment to the Water Act 1957, making it perfectly clear that the functions and powers of the Commission extend to the fluoridation of communal water supplies, when and as directed by the Minister, and also making it clear that they apply to all subordinate or derivative water authorities of whatever nature the general machinery and financial provisions of the Act are adequate.

937. One matter that will need specific attention by the draftsman is the provisions of Sec. 186 of the Local Government Act 1962. It should be made clear that if Parliament decides upon fluoridation the section has no application to works in connection with fluoridation undertaken by the Rivers and Water Supply Commission or by a municipal body or other agency at its request and direction.

938. The above indicates sufficiently the general nature of the amendments that I think would be required should Parliament decide upon fluoridation. Their detail is a matter for the draftsman. I am not called upon to advise on the amendments that may be necessary to give effect to any decision adopting any alternative to water fluoridation, but they would raise no problem of difficulty.

PART O–SUMMARY

CHAPTER 55. SUMMARY OF FINDINGS AND RECOMMENDATIONS

939. This chapter is an attempt to summarise the principal findings and recommendations of my Report. It suffers from the defect of all summaries, viz., that in the interests of brevity it omits many matters of discussion, of qualification and even of modification affecting the conclusions which in abbreviated form it seeks to reproduce. It is not in any real sense a *précis* of the full report and in case of any apparent or suspected conflict between the findings and conclusions here briefly stated and the full text of the relevant portion of the Report to which they relate, the later is to be preferred.

940. Fluoridation is the adjustment of the fluoride level of drinking water to a value recognised as optimal for the prevention of caries. In temperate climates this may be taken to approximate one part of fluoride to one million parts of water.

(Ch. 5, Ch. 17, secs (a) and (b))

941. Fluoridation is not concerned with elemental fluorine but with the fluoride ion obtained by dissolving an inorganic compound or salt of fluorine in water.

(Ch. 6, paras 39-40, 53-56)

942. The properties of the fluoride ion differ from those of elemental fluorine and its organic compounds. The latter have no direct relevance to fluoridation.

(Ch. 6, paras 42-51, 54 and 57; Chs 7 and 42)

943. Though fluorine is a member of the sub-group of elements known as halogens it differs from them in many ways and there are no relevant analogies between the physiological effects of the fluoride ion and those of other halogens, their compounds and ions.

(Ch. 6, para. 42; Ch. 34)

944. The source of the fluoride ion, i.e., the compound from which it is derived, is immaterial. The ion is the same and has the same properties irrespective of the salt or compound from which it is derived. In particular there are no chemical, physiological or other material differences between fluoride ion naturally in the water and that added artificially.

(Ch. 6, para. 55; Ch. 16, para. 292; Ch. 29, para. 534)

945. Fluorides are abundant in nature and are a significant feature of the human environment to the extent that it is doubtful that air, water, vegetation or food materials completely free from fluoride can be found.

(Ch. 7)

946. However Tasmanian drinking waters are relatively, even notably, low in fluoride content.

(Ch. 7, paras 68-71)

947. Fluoride is a normal constituent of the human body and has at least a beneficial role in human physiology. In addition the preponderance of evidence points to it as possessing an essential role; vital to skeletal and dental health.

(Ch. 8)

948. The fluoride ion is readily absorbed in the human body. Distributed in the body's fluids it is taken up and retained by the bones and teeth. It is concentrated prior to excretion in the kidneys but not retained or stored in significant quantities elsewhere. The soft tissues of the body maintain levels reflecting generally that of the blood plasma. The latter maintains a constant low level of fluoride at all but pathological levels of intake.

(Ch. 9, paras 93-100, 102-111, 138-140; Ch. 33)

949. The bones, teeth and other areas of calcification within the body attract and retain fluoride. In general the fluoride content of the skeleton will show a consistent relationship with age and with the dietary fluoride level. In this respect the level of the drinking water is the most important element. The rate of uptake will be highest during bone formation and growth and will diminish but not cease with age so long as dietary levels are maintained.

(Ch. 9, paras 112-121)

950. The teeth take up fluoride in much the same way as bone but there are differences in the absence of any continuing cellular activity in the enamel in particular. As with bone there is a consistent relationship between the level of fluoride in the drinking water, the age of the subject and the fluoride content of the teeth.

(Ch. 9, paras 122-124; Ch. 10, para. 152)

951. Fluoride may be taken up by bones and teeth in two ways. It may be incorporated into the physical structure by cellular activity during formation in which case it is said to be taken up systemically or by accretion. Or it may be taken up topically, i.e., by ion exchange at the crystal surface from the body or oral fluids. In the case of teeth, uptake by accretion is not significant after childhood and further augmentation of the fluoride content of the teeth, and the enamel in particular, is dependent on topical uptake.

(Ch. 9, paras 125-132, 134)

952. The effect of the fluoride ion when incorporated into the teeth is to enhance calcification, to provide stronger and more resistant enamel and favourably to influence the shape, structure and appearance of the teeth.

(Ch. 9, para. 136)

953. Fluoride in the oral fluids is also taken up and retained by the dental plaque; a mucoid film adhering to tooth surfaces. The fluoride content of the plaque is high. It too would seem to bear a relationship to the fluoride level of the drinking water and it has an important role in caries control.

(Ch. 10, paras 155 and 156; Ch. 11, paras 167-170)

954. The result of dental caries is demineralisation and destruction of tooth substance. Fluoride promotes host resistance by—

(*a*) Strengthening the enamel crystal and making it more resistant to acid attack.

(*b*) Inhibiting the biochemical cycle involved in the bacterial production of the attacking acid.

(*c*) Promoting remineralisation and thus retarding the advance of carious lesions.

(Ch. 9, para. 136; Ch. 11, paras 163-170)

955. Dietary discipline may contribute significantly to caries freedom in individuals by removing the carbohydrate substrate or base for caries attack. It does little or nothing to promote host resistance. In relation to the latter nothing has proved as effective as fluoride.

(Ch. 11, paras 171-177)

956. Caries, gross in degree, is prevalent in the Tasmanian community. Such data as exist enabling comparisons to be drawn would indicate that it, as a disease of childhood, is possibly even more prevalent in this State than in other Australian States. It is progressive and irreversible and in this State leads to a quite disproportionate number of young Tasmanians being either wholly or partially edentulous by the time they enter adult life. In itself and in its effects it ranks as a grave problem of public health with which existing resources in dental manpower is inadequate to cope.

(Ch. 13, paras 196-220)

957. Fluoridation at optimum levels will act to reduce the prevalence of caries in the community. The evidence substantiating this claim is overwhelming and cannot be doubted. It consists of:—

(*a*) The elucidation of chemical and physological mechanisms whereby fluoride is incorporated in tooth enamel and the description of its effects in promoting resistance to caries.

(Ch. 9, secs (f) and (g); Ch. 11, sec. (c))

(*b*) The epidemiological observations and researches of Dean and others as to the relative freedom from caries enjoyed by communities drinking naturally fluoridated water.

(Ch. 15, secs (a), (b), (c), and (d))

(*c*) The evidence of benefit furnished by those communities in which for periods up to 25 years artificial fluoridation has been practised as a deliberate community measure of caries control.

(Ch. 16, secs (a), (c), (d), (e), and paras 293-295)

958. It is also confirmed by clinical observation and experience.

(Ch. 12, para. 190; Ch. 14, paras 221-224)

959. Criticisms of fluoridation trials by Sutton and others, while indicating some methodological errors in individual instances, are unavailing to rebut the overwhelming cumulative weight of the evidence furnished both by the trials which he criticises and by subsequent confirmatory trials and clinical studies conducted elsewhere.

(Ch. 12, sec. (b); Ch. 15, paras 239-244; Ch. 16, sec. (b))

960. The extent of the benefit to be anticipated from community fluoridation will be of the order forecast by Arnold in 1943, i.e., amongst other benefits, it will lessen the prevalence of caries among 12- to 14-year-old children by a figure which will approximate 60%.

(Ch. 16, sec. (f))

961. Fluoridation is practised by communities numbering 62 million people in the United States alone. Inquiries made as to its world-wide status would seem to indicate that while opposition exists, it is gaining ground rather than losing it.

(Ch. 16, sec. (g) and Appendix 'H')

962. The epidemiological and experimental work referred to has suggested 1 ppm as the optimum level at which water should be fluoridated. Later and more exact studies have suggested slight modifications to accord with climatic and dietetic factors.

(Ch. 17, sec. (b))

963. Fluoridation at 1 ppm has been essayed in some parts of Tasmania. Notwithstanding that there is some reason to suggest that this may be low in relation to prevailing temperatures, changes in level are not recommended for the present.

(Ch. 17, sec. (c))

964. With water fluoridated at optimum levels there is a probability that up to 10% of young children will be affected by dental fluorosis or mottling due to variable water intake. No more than 2% will be of the 'mild' variety, the remainder being 'very mild' or 'questionable'. None will be noticeable except to the clinical observer. Such teeth will be vital and caries resistant. Over the child population as a whole this will be more than offset by a greater decrease in mottling and enamel defects due to idiopathic causes unconnected with fluoride. Susceptibility to dental fluorosis does not persist beyond the ninth birthday. Adults and older children are not susceptible no matter what the intake.

(Ch. 17, paras 296-297, 310-312; Ch. 20, paras 338 and 339; Ch. 21, secs (a), (b), (c), (d), (e), and (f))

965. Toxic levels of fluoride ingestion have been considered. As nothing approaching lethal levels or levels required to produce symptoms of acute toxicity could be achieved by the use of water fluoridated at 1 ppm they are not considered to be of any direct relevance.

(Ch. 19)

966. Chronic toxicity or fluorosis may result from too high levels of fluoride ingestion. Its earliest symptom is dental fluorosis or mottling in the young, and the prevalence and degree of this condition can constitute an early community warning in relation to fluoridation levels. There are many other causes of dental mottling and enamel defects in children's teeth and fluoridation at optimum levels may be expected to substantially reduce their prevalence.

(Ch. 17, para. 312; Ch. 20, paras 337-339; Ch. 21, secs (a) and (d))

967. In adults chronic toxicity or fluorosis may be manifest by—

(*a*) a symptomless osteosclerosis; or

(*b*) skeletal disabilities with, in extreme cases, neurological symptoms due to cord compression.

(Ch. 22, sec. (a))

968. With fluoridation at 1 ppm there is no reasonable apprehension of skeletal fluorosis of any kind. The threshold level above which symptomless sclerosis may occur as a result of prolonged ingestion of fluoridated water is certainly above 4 ppm and probably not lower than 8 ppm prolonged for many years. To produce disabling symptoms it is probably necessary to reach a dosage level of 20 mg a day or more for at least 10 but more probably 20 years.

(Ch. 22, secs (a), (b), (c), (d), and (e); Ch. 23, para. 427)

969. These conclusions are not vitiated by studies of native populations which under conditions of temperature diet and social habit not applicable to this community have been reported to have exhibited fluorotic symptoms at fluoride levels lower than those stated above.

(Ch. 22, secs (c) and (d))

970. In a temperate climate fluoridation at 1 ppm plainly constitutes no danger to normal individuals under ordinary social and environmental conditions. Intake may be significantly augmented in the case of some individuals due to abnormal dietary habits or industrial conditions. As far as diet is concerned there is no likelihood of harmful consequences even in a tea-drinking community, except that children under eight years of age who habitually consume seven large cups of tea a day, would, if the tea were made with fluoridated water, probably incur a 2% chance of 'moderate' dental fluorosis with brown staining.

(Ch. 22, para. 391; Chs. 26, 27, and 28)

971. Unregulated industrial exposure could be a matter of concern in some industries. The answer is industrial, and the remedy should be to control the conditions which furnish the main source of exposure. There are two Tasmanian industries in respect of which further investigation is warranted.

(Ch. 27)

972. There is just a possibility that some individuals may be found who will react abnormally to fluoride at levels far below those which will affect normal beings. The possibility is very remote but it cannot be dismissed. If such people exist and their existence has not been clearly demonstrated, the consequences are not serious.

(Ch. 24, secs (a), (b), (e), (f), (g), and (h))

973. Fluoridation at 1 ppm is not known to aggravate, dispose to or be a cause of—

(*a*) Diabetes.
(Ch. 25, sec. (a))

(*b*) Kidney disease.
(Ch. 9, secs (c) and (h); Ch. 25, sec. (b); and Ch. 33)

(*c*) Cancer.
(Ch. 32)

(*d*) Goitre.
(Ch. 34)

(*e*) Diseases of the cardio-vascular system.
(Ch. 36)

(*f*) Periodontal disease.
(Ch. 31)

(*g*) Enzymatic malfunction.
(Ch. 36)

(*h*) Teratogenism and disorders of pregnancy or childbirth.
(Ch. 37)

(*i*) Mongolism.
(Ch. 38)

(*j*) Ectopic calcification within the human body.
(Ch. 39)

(And see generally Ch. 29, para. 537 and Ch. 30)

974. Comparative studies of vital and other statistics for fluoridated and unfluoridated communities do not support any suggestion that fluoridation has an adverse effect on morbidity or mortality. The great preponderance of evidence is to the contrary.

(Ch. 22, sec. (b); Ch. 30)

975. The effects of fluoridation are not determined or modified in any material way by the hardness or softness of the drinking water. Fluoridation does not deprive the body of calcium or affect, other than beneficially, calcium balance within the body.

(Ch. 9, para. 135; Ch. 40; and Ch. 41)

976. There is no risk of any significant supplement to dietary fluoride resulting from the ingestion of organic compounds of fluorine such as may occur in fluorinated drugs. The use of inorganic fluoride compounds for pharmaceutical purposes should be regulated, and these precautions taken, there should be no risk of deleterious results.

(Ch. 42)

977. Fluoridation does not affect the palatability, taste or smell of communal water supplies. It poses no community problems in agriculture, animal husbandry, horticulture or in cooking and food processing.

(Chs 46, 47, and 48)

978. There is no reasonable ground for apprehending a build-up of fluoride in reticulation systems or of overdosage by accident or design. Water fluoridated at 1 ppm is not corrosive.

(Ch. 49, secs (a), (b), and (c))

979. The maximum dental benefit to be gained from drinking fluoridated water will accrue during infancy and childhood. However, there is an adult benefit to be gained from continuing ingestion. In addition to dental benefit there is a strong probability that other physiological benefits will come to be confirmed in relation to osteoporosis and calcification of the aorta.

(Ch. 43, secs (a), (b), (c), and (d))

980. Fluoridation of communal water supplies has inescapable consequences for all members of the community. However, fluoridation is designed to benefit children. This is a factor of preponderating weight in any argument that seeks to deny the community's right to introduce it on the ground that it is an interference with personal freedom.

(Ch. 44, sec. (b))

981. Fluoridation does not infringe religious freedom.

(Ch. 44, sec. (c))

982. Fluoridation is not unreasonably described by its opponents as 'mass prophylaxis' or even—though possibly inaccurately—as 'mass medication'. But its propriety should be determined on its intrinsic merits, not on semantic labels.

(Ch. 44, sec. (d))

983. Fluoridation does not involve a breach of any rule of medical ethics or professional propriety.

(Ch. 44, sec. (e))

984. There are no valid analogies either in support of or in opposition to fluoridation to be found in other public health measures such as vaccination, chlorination, etc. It is a measure *sui generis*. It will constitute a precedent in the sense of providing a reference point in future discussions of public health matters but in no way could it be said to be a precedent in a legal or constitutional sense. In the absence of any binding force, no weight is attributed to the argument that fluoridation may constitute a precedent.

(Ch. 44, sec. (e))

985. Whether or not to fluoridate communal water supplies requires a decision on a major matter of public health and the decision should be made by Parliament. It is not a matter of water supply for water purposes and to regard it as such is to confuse the principle with the vehicle of administration. The decision required transcends the capacity of local government. It is not a suitable subject to be decided by popular referendum and in any case to entrust the decision of such a serious matter of public health to a referendum would be an abrogation of Parliamentary responsibility.

(Ch. 51)

986. As a public health measure, no alternative means of adding a fluoride supplement to the communal diet is as efficacious as the fluoridation of water supplies. The issue of tablets is not a satisfactory substitute. Even less satisfactory would be fluoridated milk. If an alternative is required, fluoridated salt offers the most promise, but many details of its application have still to be worked out and it should not be expected to confer the same degree of benefit as water fluoridation.

(Ch. 52, secs (a), (b), (c), (d), and (e))

987. Fluoridation is cheapest when put into effect in connection with large regional schemes of water treatment or supply. Under such circumstances it should not cost more than 20 cents per capita per annum. It is dearest when brought into operation in small communities with scattered water inlets and lacking other water treatment plant, staff and equipment. Within these extremes there is such a wide range of possibilities that no certain estimate of costs can be given.

(Ch. 53)

988. Because of the disparity of public benefit no true balance can be struck between the efficacy of water fluoridation and the cost of a fluoride tablet programme even if it be cheaper.

(Ch. 53, sec. (c))

989. If fluoridation be decided upon by Parliament, then the present Fluoride Committee should be reconstituted and its functions redefined. It should be responsible for advisory and supervisory functions in relation to fluoridation but the implementation of any particular scheme should, under Ministerial direction, be the responsibility of the Rivers and Water Supply Commission. If fluoridation be left in its present position as a permissive measure to be adopted by municipalities as they see fit, then there would seem to be little point in seeking any amendments to existing statutory provisions.

(Ch. 54)

APPENDIX 'A'

LIST OF PERSONS WHO APPEARED AND GAVE EVIDENCE, EITHER ON THEIR OWN BEHALF, OR ON BEHALF OF THE ORGANISATIONS NAMED

Witness	
Dr N. D. Abbott, Hobart	Medical Practitioner, Member of the House of Assembly, Tasmania
Sir Arthur Amies, South Yarra, Victoria	Professor of Dental Medicine and Surgery, and Dean of the Faculty of Dental Science at the University of Melbourne
Dr P. W. Arkle, North Hobart	Senior Dental Officer of the Department of Health Services, Tasmania
Mr J. D. Arnold, Gravelly Beach	Waterworks Supervisor for the Municipality of Beaconsfield
Professor H. F. Atkinson, Heidelberg, Victoria	Professor of Dental Prosthetics at the University of Melbourne
Professor A. G. Baikie, Hobart	Professor of Medicine at the University of Tasmania
Mr A. R. Blenkhorn, Railton	Proprietor, Railton Lime Works
Mr R. C. Bradley, Beaconsfield	Pharmaceutical Chemist
Dr V. G. Bristow, Toorak, Victoria	Medical practitioner and physician in charge of the Allergy Clinic at the Alfred Hospital, Melbourne
Dr (DENT.) P. C. Brothers, Sandy Bay	Dental practitioner and Consultant to Government of Tasmania on Fluoridation and Deputy Assistant Director of Dental Services, Dept of the Army, Tasmania
Mr R. L. J. Brough, Scottsdale	Council Clerk of the Scottsdale Municipal Council
Mr B. H. Brown, Blackman's Bay	Retired civil servant
Mr J. A. Brown, Lindisfarne	Fluoridation Technician for the Hobart City Council
Mr W. E. Bull, Beaconsfield	Farmer
Dr (DENT.) T. E. Canning, Hobart	Dental Practitioner
Dr H. R. Cash, Ivanhoe, Victoria	Dental Officer, Melbourne City Council
Mr E. C. Coleman, New Town	Executive Officer, Department of Health Services, Hobart
Professor V. L. Collins, Hawthorn, Victoria	Professor of Paediatrics at the University of Melbourne
Sir Robert Cosgrove, West Hobart	President of the Denison Branch of the Australian Labor Party
Mrs J. H. Coy, Hobart	Nutrition Officer of the Department of Health Services, Tasmania
Mr P. G. Crawford, Sandy Bay	City Engineer, City of Hobart
Mr T. W. Davenport, Ulverstone	Public Servant
Mr J. E. Dean, Berriedale	Town Clerk, City of Glenorchy
Mr W. D. Dickens, Lindisfarne	Committee on Publication, First Church of Christ Scientist, Hobart
Mr M. G. Doyle, Taroona	Council Clerk of the Kingborough Municipal Commission
Mr D. G. Dudgeon, Howrah	Council Clerk of the Clarence Municipal Commission
Mrs C. E. N. Dwyer, West Hobart	Housewife
Dr. (DENT.) A. D. Eslake, Smithton	Government District Dental Officer
Dr M. J. Flynn, Roseville, N.S.W.	Chief Medical Officer of the Metropolitan Water, Sewerage and Drainage Board, Sydney
Dr P. D. Grant, Beaconsfield	Medical Practitioner
Mr G. H. Gray, Rockhampton, Queensland	Member of the House of Representatives, Commonwealth of Australia. President of the Anti-Water Fluoridation Council of Australia and New Zealand
Mr F. J. Grey, West Riverside	Chemist-in-Charge of the West Tamar Water Supply Filtration Plant
Dr E. T. Hale, Howrah	Medical practitioner and President of the Tasmanian Pure Water Association
Mr K. V. Harris, Taroona	Engineer for Development, Hobart City Council
Mr S. C. G. Hawker, Sandy Bay	Commissioner, Kingborough Municipal Commission
Mr A. J. Hepworth, Springfield	Water Supply Maintenance Overseer, Hobart City Council
Professor E. S. Holdsworth, Taroona	Professor of Biochemistry at the University of Tasmania
Mr W. A. Hurle, Lenah Valley	Town Clerk, City of Hobart
Mr R. A. Illingsworth, Beaconsfield	Pensioner
Mr L. V. Jacques, Scottsdale	Headmaster, Scottsdale District School
Mr L. J. Kausman, Hawthorn, Victoria	Pharmaceutical Chemist and Secretary of the Victorian Anti-Fluoridation Association
Mr K. F. Lowrie, New Town	Mayor, City of Glenorchy
Sister Luke, Beaconsfield	Sister-in-Charge, St Francis Xavier School, Beaconsfield
Dr J. R. Macintyre, Taroona	Deputy Director-General of Health Services, Tasmania
Mr R. D. Mainwaring, Lindisfarne	Chairman, Clarence Municipal Commission
Mr J. D. Manzoni, Beauty Point	Council Clerk of the Beaconsfield Municipal Council
Dr (DENT.) H. R. J. Marshall, Taroona	Practising Dentist. President Australian Dental Association (Tasmanian Branch)
Professor N. D. Martin, Wahroonga, N.S.W.	Professor of Preventive Dentistry at the University of Sydney
Dr A. Mcl. Millar, Hobart	Medical practitioner and Spokesman, Australian Medical Association (Tasmanian Branch)
Dr. T A. O'Donnell, Burwood, Victoria	Acting Professor, Department of Inorganic Chemistry at the University of Melbourne
Mr R. B. Pears, Lenah Valley	Water Superintendent, Hobart City Council

APPENDIX 'A'—*continued*

Witness	
Dr J. B. Polya, Mt Stuart	Associate Professor and Dean of Faculty of Science at the University of Tasmania
Dr (DENT.) D. G. Ramsden, Bridport	Government District Dental Officer for Scottsdale Dental District
Mr V. C. Richardson, Ulverstone	Warden, Ulverstone Municipal Council
Dr A. D. Ross, Taroona	Director of Public Health, Tasmania
Mr E. A. Rowe, Berriedale	Chairman, Rivers & Water Supply Commission, and Metropolitan Water Board, Tasmania
Mr H. V. Salier, Scottsdale	Warden, Scottsdale Municipal Council
Mr D. G. Schaffner, Sandy Bay	Engineering Member, Rivers & Water Supply Commission, Tasmania
Mr M. R. Shipp, Sandy Bay	Government Analyst and Chemist, Tasmania
Mr J. H. Skinner, Huonville	Manager, Bulk Oil Depot and Councillor, Huon Municipal Council
Dr (DENT.) A. H. Stabler, Beaconsfield	Government District Dental Officer
Dr A. Stoller, St Kilda, Victoria	Director of Mental Health Research Institute of the Mental Health Authority in Victoria
Professor E. Storey, Kew, Victoria	Professor of Conservative Dentistry at the University of Melbourne
Mr A. G. Strom, Sandy Bay	Consulting Engineer, Gutteridge, Haskins & Davey, Hobart
Mr F. J. Strong, George Town	Technical Services Manager of Comalco Aluminium (Bell Bay) Ltd
Dr P. R. N. Sutton, Middle Brighton, Victoria	Senior Lecturer in Dental Science at the University of Melbourne
Professor J. C. Thonard, Kensington Park, South Australia	Professor of Dental Science at the University of Adelaide
Mr H. W. Viney, Burnie	Farmer
Mr F. R. Watson, Beaconsfield	Headmaster, Beaconsfield Area School
Mr A. Webb, Invermay	Pensioner
Mr W. D. Wivell, Sidmouth	Warden, Beaconsfield Municipal Council
Mr V. G. Yaxley, Burnie	Council Clerk, Burnie Municipal Council
Mr W. T. Young, Stowport	Warden, Burnie Municipal Council

All Witnesses are residents of Tasmania, except where otherwise stated.

APPENDIX 'B'

LIST OF PERSONS WHO TENDERED SUBMISSIONS, FORWARDED INFORMATION OR CORRESPONDED WITH THE COMMISSION BUT WHO DID NOT GIVE EVIDENCE

*Persons who tendered submissions are shown thus**

Adamson, Dr K., CMG, Melbourne, Victoria	President, 18th Australian Dental Congress
Allen, Mr C. A., Launceston, Tasmania	Town Clerk, City of Launceston
Allison, Mr N. S., Devonport, Tasmania	Council Clerk, Municipality of Devonport
Ambassade De Belgique, Canberra, A.C.T.	
Ambassade De France, Canberra, A.C.T.	
Anderson, Mr G., Sydney, N.S.W.	Manager, Dental Health Products
Andrews, Dr N. H., Melbourne, Victoria	Director of Child Health, (Dental) Victoria
Aslander, Dr A., Sweden	Retired
Austrian Embassy, Canberra, A.C.T.	
Bailey, Mr V. L., Hamilton, Tasmania	Council Clerk, Municipality of Hamilton
Baker, Mr R. D., Hobart, Tasmania	Managing Director, Bakers Milk (Hobart) Pty Ltd
Barics, Mr B., Tullamarine, Victoria	
Barnard, Mr P. D., Sydney, N.S.W.	Senior Lecturer, Department of Preventive Dentistry, University of Sydney
Beaney, Mr H. L., Adelaide, S.A.	Director and Engineer-in-Chief, Engineering and Water Supply Department, South Australia
Beck, Mr D. J., Dunedin, N.Z.	Senior Lecturer in Charge, Department of Preventive Dentistry, University of Otago Dental School
Beekmeijer, Mr J. F., Derby, Tasmania	Council Clerk, Municipality of Ringarooma
Bell, Dr Muriel E., CBE, Dunedin, N.Z.	Retired
Bond, Mr H. G., Hobart, Tasmania	Regional Director, Bureau of Meteorology
Bonney, Mr C., Perth, W.A.	Head of Department of Dental Health Education, Perth Dental Hospital
British High Commission, Canberra, A.C.T.	
*Brown, Mr J. H., North Hobart, Tasmania	Retired
Bruce, Mr W., Melbourne, Victoria	First Church of Christ Scientist
Burgstahler, Professor A. W., Kansas, U.S.A.	Professor of Chemistry, University of Kansas
*Burlison, Mr G., North Clayton, Victoria	
*Butterworth, Mr R. W., Richmond, Tasmania	Council Clerk, Municipality of Richmond
Cahill, Hon. C. J., MLC, Sydney, N.S.W.	Member of Parliament
Campbell, Dr E. M., Bethesda, U.S.A.	Acting Chief Disease Control Branch, Division of Dental Health, U.S. Department of Health Education and Welfare
Candau, Dr M. G., Geneva, Switzerland	Director-General, World Health Organisation
Carey, Professor S. W., Hobart, Tasmania	Professor of Geology, University of Tasmania
Carr, Mr L. M., Canberra, A.C.T.	Senior Dental Officer, Commonwealth Department of Health
Chapman, Mr L. E. H., Stroud, N.S.W.	Councillor, Stroud Shire
Clayton, Mr H. A., Westbury, Tasmania	Council Clerk, Municipality of Westbury
Clements, Dr F. W., Sydney, N.S.W.	School of Public Health and Tropical Medicine, University of Sydney
Consul General of the Polish Peoples' Republic, Sydney N.S.W.	
Consulado De Equador, Melbourne, Victoria	
Consulado General De Chile, Sydney, N.S.W.	
Consulado General De Colombia, Sydney, N.S.W.	
Consulado General De Panama, Sydney, N.S.W.	
Consulat De Monaco, Melbourne, Victoria	
Consulate for Liberia, Adelaide, S.A.	
Consulate General of Guatemala, Sydney, N.S.W.	
Consulate General of Iceland, Melbourne, Victoria	
Consulate General of the Czechoslovak Socialist Republic Sydney, N.S.W.	
Consulate General of the Peoples' Republic of Bulgaria, Sydney, N.S.W.	
Consulate of the Dominican Republic, Melbourne, Victoria	
Consulate of the Republic of Venezuela, Melbourne, Victoria	
Creighton, Mr R. M., Artarmon, N.S.W.	Managing Director, R. M. Creighton Pty Ltd
Cross, Dr D. O., Sydney, N.S.W.	Honorary Secretary, Section of Allergy, A.M.A., N.S.W. Branch
Cunningham Dax, Dr E., Melbourne, Victoria	Director, Mental Health Authority, Victoria
Davies, Professor G. N., Brisbane, Queensland	Dean, Dental College, University of Queensland
Davies, Mr R. B., Hobart, Tasmania	
Deegan, Mr A. K., Queenstown, Tasmania	Council Clerk, Municipality of Queenstown
Department of Health, Pretoria, South Africa	
Dietzius, Mr J. F., Mona Vale, N.S.W.	
*Dobbs, Dr C. B., Bangor, Wales	Senior Lecturer (Mycology) University College of North Wales

APPENDIX 'B'—*continued*

*Donnelly, Mr R. P., Claremont, W. A.	Chartered Chemical Engineer
Downes, Mr H. E., Canberra, A.C.T.	Acting Director-General of Health, Commonwealth Department of Health
Dunne, Mr T. C., Perth, W.A.	Director of Agriculture, Western Australia
Dunning, Mr A. W., George Town, Tasmania	Council Clerk, Municipality of George Town
Eastman, Mr M. F., Alonnah	Council Clerk, Municipality of Bruny
Ellis, Mr A. L.	General Manager, Comalco Aluminium (Bell Bay) Ltd Tasmania
Embaixada De Portugal, Canberra, A.C.T.	
Embajada De La Republica Argentina, Canberra, A.C.T.	
Embassy of Brazil, Canberra, A.C.T.	
Embassy of Ireland, Canberra, A.C.T.	
Embassy of Japan, Canberra, A.C.T.	
Embassy of Switzerland in Australia, Canberra, A.C.T.	
Embassy of the Federal Republic of Germany, Canberra, A.C.T.	
Embassy of the Philippines, Canberra, A.C.T.	
Embassy of the Socialist Federal Republic of Yugoslavia, Canberra, A.C.T.	
Embassy of the Union of Burma, Canberra, A.C.T.	
Embassy of the Union of Soviet Socialist Republics, Canberra, A.C.T.	
Embassy of the United Arab Republic, Canberra, A.C.T.	
Enright, Mr A. J., Deloraine, Tasmania	Council Clerk, Municipality of Deloraine
Evans, Mr W. J., Deewhy, N.S.W.	Regional Returning Officer for the Electorate of McKellar, N.S.W.
Ewing, Professor Sir Maurice, Melbourne, Victoria	Professor of Surgery, Royal Melbourne Hospital
*Exner, Dr F. B., Seattle, U.S.A.	Medical Practitioner
Falconer, Mr J. R., Smithton, Tasmania	Council Clerk, Municipality of Circular Head
Fanning, Dr Elizabeth A., Adelaide, S.A.	Reader in Preventive Dentistry, University of Adelaide
Farnbach, Dr R. J., Melbourne, Victoria	Chief Health Officer, Commission of Public Health, Victoria
*Fink, Mr V., Sydney, N.S.W.	Assistant Secretary, Citizens Safe Water Committee
Finnish Legation, Canberra, A.C.T.	
Forno, Mr A. H., Cairns, Queensland	Shire Clerk, Mulgrave Shire Council
Fremlin, Professor J. F., Birmingham, England	Professor of Applied Radio-activity, The University of Birmingham
Fuller, Col J. Ferris, Wellington, N.Z.	Retired
*Fulton, Mrs D. A., West Launceston, Tasmania	Housewife
*Fulton, Mr R., Toronto, N.S.W.	Secretary, Hunter District Anti-Fluoridation Association
Gane, Mr D. I., Ulverstone, Tasmania	Council Clerk, Municipality of Ulverstone
Garwood, Mr B. M., Evandale, Tasmania	Council Clerk, Municipality of Evandale
Gibson, Mr J., Sheffield, Tasmania	Council Clerk, Municipality of Kentish
*Gliddon, Mrs N., Darlington, W.A.	Housewife
Grainger, Mr W. A., Sydney, N.S.W.	Federal President, Australian Dental Association
Gray, Mr M. N. F., Devonport, Tasmania	Manager, Gordon Edgell Pty Ltd
Grove, Dr J. L., Launceston, Tasmania	Medical Practitioner
Gurr, Mr A. K., St Leonards, Tasmania	Council Clerk, Municipality of St Leonards
Hall, Mr G.	General Superintendent, Electrolytic Zinc Coy of Australasia Ltd
Halton, Mr C. J., Oatlands, Tasmania	Council Clerk, Municipality of Oatlands
*Harding, Mr J. E., Rockhampton, Queensland	Publicity Officer, Anti-Fluoridation Council of Australia and New Zealand
Harding, Mr W. B., Tamworth, N.S.W.	Veterinary Officer, Tamworth Pastures Protection Board
Harris, Mr R. John, Hobart, Tasmania	Consul for Sweden in Tasmania
Hartam, Mr L., Port Arthur, Tasmania	Council Clerk, Municipality of Tasman
*Hinks, Mr A. C., Ryde, N.S.W.	Hon. Secretary, Registrar, Australian Naturopathic Physicians Association Ltd
Hodgson, Mr H. J. N., Adelaide, S.A.	Retired
Homes, Mr K. E., Claremont, Tasmania	Chief Chemist, Cadbury-Fry-Pascall Pty Ltd
*Horsnell, Professor A. M., Adelaide, S.A.	Professor of Dental Science, University of Adelaide
Imperial Ethiopian Consulate General, Sydney, N.S.W.	
*Jaffray, Mr K. S., Townsville, Queensland	Naturopath
Jamieson, Mr G., Latrobe, Tasmania	Council Clerk, Municipality of Latrobe
*Jansen, Miss Isabel, Antigo, Win. U.S.A.	Registered Nurse
Johnson, Mr B. R., Bothwell, Tasmania	Council Clerk, Municipality of Bothwell
Johnson, Dr D. W., Brisbane, Queensland	Deputy Director-General of Health and Medical Services, Queensland
Johnson, Mr T. G., Ross, Tasmania	Council Clerk, Municipality of Ross
*Joll, Mr A., E. Hawkley, Liss. Hampshire, England	Chairman, National Pure Water Association
Jones, Mr H. V., Waratah, Tasmania	Council Clerk, Municipality of Waratah
Kearnan, Mr J. R., Adelaide, S.A.	Crown Solicitor, South Australia
Kearney, Mr D. A., Hobart, Tasmania	Registrar, University of Tasmania
Kelly, Mrs E., Hobart, Tasmania	Hon. Secretary, Southern Tasmanian Council of School Mothers' Clubs

APPENDIX 'B'—*continued*

*Kelly, Mr H. H., Richmond, Tasmania	Retired
Kellett, Mr H., New Town, Tasmania	Retired
Keys, Mr M. E., Tamworth, N.S.W.	Town Clerk, City of Tamworth
Knight, Mr G. V., Huonville, Tasmania	Council Clerk, Municipality of Huon
*Krueger, Mr V. A., Albury, N.S.W.	
Laird, Mr D., Triabunna, Tasmania	Council Clerk, Municipality of Spring Bay
Lamsvelt, Dr G. A., Netherlands	Head of the Division for International Health Affairs
Latvian Consulate in Melbourne, Melbourne, Victoria	
Lazar, Mr P. F., Sydney, N.S.W.	Director, Dental Health Education and Research Foundation, N.S.W.
Long, Mr H. C., Monrovia, California, U.S.A.	Vice President, The National Health Federation
Longley, Mr E. O., Sydney, N.S.W.	Acting Director, Division of Occupational Health, Department of Public Health, N.S.W.
Ludwig, Dr T. G., Wellington, N.Z.	Director, Dental Research Unit, The New Zealand Medical Research Council
Mackay-Smith, Dr G., Hobart, Tasmania	General Superintendent, Royal Hobart Hospital
Mallon, Mr R. F., Sydney, N.S.W.	Commonwealth Electoral Officer for N.S.W.
Malta High Commission, Canberra, A.C.T.	
Marriott, Mr R. L., Geeveston, Tasmania	Council Clerk, Municipality of Esperance
Mathieson, Dr J. B., Canberra, A.C.T.	Secretary, National Health and Medical Research Council
McDonnell, Cathleen, Perth, W.A.	Hon. Secretary, Pure Water Association (West Australian Branch)
McFie, Mr D. G., Hobart, Tasmania	Registered Dental Mechanic
McKenna, Mr J. T., Penguin, Tasmania	Council Clerk, Municipality of Penguin
McLeod, Mr H. S., New Norfolk, Tasmania	Council Clerk, Municipality of New Norfolk
McMillan, Mr D. F., Wellington, N.Z.	Division of Public Health, New Zealand
*McNeill, Mr L. E., Campbell Town, Tasmania	Council Clerk, Municipality of Campbell Town
McPherson, Mr K. D., Moonah, Tasmania	Estate Agent
Menz, Mr G., Sydney, N.S.W.	Assistant Crown Solicitor, N.S.W.
Meyers, Dr E. S. P., Sydney, N.S.W.	Director of State Health Services, N.S.W. and Chairman, Fluoridation of Public Waters Advisory Committee
*Mick, Dr R. J. H., Treasure Island, Florida, U.S.A.	Dental Practitioner
Mildern, Florence L., Sydney, N.S.W.	Dental Health Educator, Dental Health Education and Research Foundation, N.S.W.
Miller, Mr L. W., Hobart, Tasmania	Chairman, Pesticides Board
*Millington, Mrs D. M., New Town, Tasmania	Housewife
Ministry of Health, London, England	
Moon, Mrs M.E., Devonport, Tasmania	Housewife
Moore, Mr R. H., Currie, King Island	Council Clerk, Municipality of King Island
Moore, Mr W. F., Strahan, Tasmania	Council Clerk, Municipality of Strahan
Morgan, Mr J., Brookvale, N.S.W.	Shire Clerk, Warringah Shire Council
*Morrow, Mr S., Belmont, N.S.W.	President, Hunter District Anti-Fluoridation Association
Myers, Miss Ruth, Geneva, Switzerland	Scientist, Dental Health, World Health Organisation
*Newton, Mr J. M., Sydney, N.S.W.	Federal Secretary, Australian Dental Association
New Zealand High Commission, Canberra, A.C.T.	
Oakley, Mr G. R., Melbourne, Victoria	Chairman, Fluoridation Committee, Australian Dental Association (Victorian Branch)
*O'Connell, Hon. D. D. O., MLC, N.S.W.	Member of Parliament
Office of the High Commissioner for Canada, Canberra, A.C.T.	
Office of the High Commissioner for Ceylon, Canberra, A.C.T.	
Office of the High Commissioner for the Republic of Ghana, Canberra, A.C.T.	
*Pardy, Mr J. E., Forbes, N.S.W.	Refrigeration, Electrical and Automotive Engineer
Peacock, Mr D. H., Hobart, Tasmania	Production Manager, H. Jones & Co. Pty Ltd
*Penlington, Mr W. A. G., Havelock, North N.Z.	President, Hastings Anti-Fluoridation Society
Phillips, Dr C. W., Hobart, Tasmania	Commonwealth Director of Health, Tasmania
Press, Mr E. J., St Helens, Tasmania	Council Clerk, Municipality of Portland
Purves, Mr R. J., Hobart, Tasmania	Senior Superintendent, Administration, Education Department, Hobart
Refshauge, Sir William, Canberra, A.C.T.	Commonwealth Director-General of Health
Reid, Mr S. G., Brisbane, Queensland	Government Printer, Queensland
Renfree, Mr H. E., Canberra, A.C.T.	Commonwealth Crown Solicitor
Republic of Haiti, Sydney, N.S.W.	
Republica De Bolivia, Consulate General, Brisbane, Queensland	
Richard, Mr R. B., Hobart, Tasmania	Secretary, Municipal Association of Tasmania
*Robertson, Mr R. C., Newnham, Tasmania	
*Roche, Mr D., Hobart, Tasmania	Veterinary Specialist (Animal Nutrition) of Animal Health Service
Rock, Mr E., Melbourne, Victoria	Assistant National Director, Australian League of Rights
Royal Danish Embassy, Canberra, A.C.T.	
Royal Norwegian Consulate General, Sydney, N.S.W.	

APPENDIX 'B'—*continued*

Royal Swedish Embassy, Canberra, A.C.T.	
Royal Thai Embassy, Canberra, A.C.T.	
Rudko, Dr V., Geneva, Switzerland	Chief, Dental Health, World Health Organisation
Russell, Mr R. C., Babinda, Queensland	
Russell, Professor A. L., Michigan, U.S.A.	Professor, Dental Public Health, School of Public Health, University of Michigan
Ryan, Mr W. E., Brisbane, Queensland	Solicitor-General, Queensland
*Sansom, Mr C., Mt Stuart, Tasmania	
Sadlier, Mr D., Moscow, U.S.S.R.	First Secretary, Australian Embassy, Moscow
Sale, Mr J. H., Kempton, Tasmania	Council Clerk, Municipality of Green Ponds
Schrotenboer, Mr G. H., Washington, U.S.A.	Assistant Secretary, Council on Dental Therapeutics, American Dental Association
Scott, Mr R., Clayton, Victoria	Wallace and Tiernan Pty Ltd
Shea, Mr L. L., Hobart, Tasmania	Secretary, Dental Mechanics Registration Board, Tasmania
South African Embassy, Canberra, A.C.T.	
Stanley, Mr M., Dublin, Eire	Secretary, Department of Health, Eire
*Stanton-Hicks, Professor Sir C., Glen Osmond, S.A.	Retired
*Stepanoff, Mr A., Biloela, Queensland	Secretary, Biloela Pure Water Committee
*Steyn, Professor Douw G., Pretoria, Republic of South Africa	Emeritus Professor of Pharmacology, University of Pretoria
Stewart, Mr N. D., Sorell, Tasmania	Council Clerk, Municipality of Sorell
Stocker, Mr R. O. B., Sydney, N.S.W.	Managing Director, Denta-Health Products
Stone, Mr P. R., Devonport, Tasmania	Manager, A. Wander (Aust.) Pty Ltd
Strang, Mr A., Gormanston, Tasmania	Council Clerk, Municipality of Gormanston
Strutton, Mr R., Brisbane, Queensland	Under-Secretary, Department of Health, Queensland
Styles, Mr G. G., Bendigo, Victoria	Secretary, Bendigo Trades Hall Council and Literary Institute
Tapping, Mr D. W., Hobart, Tasmania	State Secretary, Federated Pharmaceutical Service Guild of Aust., (Tasmanian Branch)
*Taylor, Mr R. L., Perth, W.A.	Reader in Prosthodontia at the University of Western Australia and President, Australian Dental Association, Western Australian Branch
The Hashemite Kingdom of Jordon, Honorary Consul in Sydney, Sydney, N.S.W.	
The High Commission of India, Canberra, A.C.T.	
The Royal Embassy of Greece, Canberra, A.C.T.	
Tonkin, The Hon. J. T., MLA, Perth, W.A.	Leader of the Opposition, Western Australia
Van Der Kek, Mr A. M., Lilydale, Tasmania	Council Clerk, Municipality of Lilydale
Van Deventer, Mr B. J. S., Pretoria, South Africa	Secretary, Report of the Commission of Inquiry into Fluoridation, Republic of South Africa
*Venn, Mrs K. J., Bellerive, Tasmania	Federal Secretary, Federated Association of Australian Housewives
Viney, Mr B. M., Longford, Tasmania	Council Clerk, Municipality of Longford
Von Steiglitz, Mr D., Brighton, Tasmania	Council Clerk, Municipality of Brighton
*Waldbott, Dr G. L., Detroit, Michigan, U.S.A.	Medical Practitioner
Warner, Mr B., Scottsdale, Tasmania	Manager, Kraft Foods Limited
*Watson, Professor H. E., Woking, Surrey, England	
*Webber, Mr J. T., Sydney, N.S.W.	Hon. Secretary, Anti-Fluoridation Association of N.S.W.
Wells, Mr F. V., London, England	
Williams, Mr G. F., Zeehan, Tasmania	Council Clerk, Municipality of Zeehan
Williams, Mr L. R. A., Melbourne, Victoria	Chairman, Lecture Programme Committee, 18th Australian Dental Congress
Woodruff, Dr P. S., Adelaide, S.A.	Director-General of Public Health, South Australia
Worsley, Mr K. G., Whitemark, Tasmania	Council Clerk, Municipality of Flinders
Yates, Mr R. H., Fingal, Tasmania	Council Clerk, Municipality of Fingal
Yaxley, Mr J. D., Wynyard, Tasmania	Council Clerk, Municipality of Wynyard
Zur, Dr J., Chicago, U.S.A.	Secretary, Council on Dental Health, American Dental Association

APPENDIX 'C'*

LIST OF EXHIBITS

* This Appendix is not reprinted. See footnote to paragraph 11 of the Report.

APPENDIX 'D'

SUMMARY OF SOME REPORTS OF INQUIRIES INTO FLUORIDATION OF PUBLIC WATER SUPPLIES

Ex.	*Country*	*Date*	*Reporting Body*	*Summary of Findings*
1	United Kingdom	1952	Mission to the U.S.A. appointed by the Ministry of Health	1. Fluoridation would be useful in the United Kingdom. 2. Preliminary fluoridation surveys should be begun, and research into long term effects on humans encouraged 3. Simple alternative methods for the population not on a piped water supply should be investigated
5	United Kingdom	1962	Steering Committee appointed by the Ministry of Health	1. Five years of fluoridation at 1 ppm in 3 areas has caused a substantial improvement in the deciduous teeth of young children, in line with American experience 2. Fluoridation presents no technical difficulties and there is no evidence of harm 3. The raising of the fluoride content of drinking water to a level of 1 ppm is safe.
3	New Zealand	1957	Commission	1. Fluoridation will give benefits to dental health without any disadvantages, and is the only practicable method of supplying fluoride as a public health measure. 2. A national body should control fluoridation but local authorities should decide whether to fluoridate 3. A referendum is an unsatisfactory method of reaching a decision on fluoridation 4. Fluoridation does not interfere with personal liberties
21	World Health Organisation	1957	Expert Committee	1. Drinking water containing 1 ppm fluoride has a marked caries-preventive action with maxium benefit if consumed during life 2. There is no evidence of impairment to health from this water 3. Controlled fluoridation of drinking water is a practicable and effective public health measure
		1967	Monograph: 'Fluorides and Human Health' by International Contributors. (Ericsson, Editor) (In course of preparation. Expected to be available by end of 1968).	
22	South Africa	1960	Expert Committee appointed by Council for Scientific and Industrial Research	1. Fluoridation will reduce the ravages of dental caries and the benefits to be obtained from it outweigh any speculative disadvantages 2. There is no proof that fluoride at the recommended level causes ill effects 3. Fluoridation is now beyond the experimental stage and no specific research on it is required
1038	South Africa ..	1966	Commission	1. There are well defined limits of fluorine ingestion for adults and children which should not be exceeded 2. Fluoridation to the optimum level which according to temperature will range from 0.7 to 1.2 ppm will substantially reduce the prevalence of dental caries and will be entirely without detrimental effect on consumers 3. Fluoridation is the most suitable public health measure to utilize the advantages of fluoride
4	Ontario, Canada	1961	Committee	1. Fluoridated water at 1 ppm strikingly reduces the incidence of dental caries when consumed during the period of tooth development, without harm to bodily health 2. Fluoridation equipment is reliable and precise and fluoridated water at 1 ppm does not cause corrosion or affect industrial processes adversely 3. The cost is very reasonable in relation to the community health benefit, and no practical alternative to fluoridation is evident
525	U.S.A.	1957	Reference Committee of House of Delegates of American Medical Association	1. Fluoridated water at 1 ppm or a level appropriate to locality, decreases incidence of caries in childhood and into adulthood 2. There is no proof of harm to the general health of the population from drinking fluoridated water for long periods, and the minimal dental fluorosis associated with it is outweighed in importance by the caries inhibiting effect of the fluoride 3. Fluoridation should be regarded as a community prophylactic measure to reduce tooth decay where the water supply contains less than the optimum level of fluorine

APPENDIX 'D'—*continued*

Ex.	*Country*	*Date*	*Reporting Body*	*Summary of Findings*
550	Sweden	1958	Royal Medical Board	1. Fluoridation of drinking water at 1 ppm will reduce dental decay incidence in children and give effective protection of teeth into middle age, and there is no hazard to health 2. Objections in principle cannot be allowed to obstruct this important public health measure 3. There must be strict control and regular dental and medical examination of children and young persons in the fluoridated areas 4. Alternative vehicles for fluoride administration cannot replace water fluoridation
2	Australia (Victoria)	1954	Special Committee appointed by Commission of Public Health	1. Fluoridation of water supplies should be practised at the optimal level of 1 ppm under strict control and under trial conditions for valid comparison with other parts both medically and dentally 2. Topical application of fluoride in non-reticulated areas should be investigated and public education in prevention of dental caries pursued 3. Consideration should be given to assisting water authorities to meet the costs of fluoridation
6	Australia (New South Wales)	1963	Dr M. J. Flynn to Minister for Health	1. Fluoridation of water supplies is the best method of preventing and reducing dental decay, and is warranted in Sydney and N.S.W. 2. Ample experience in many countries shows its practicability, efficacy, safety and economy, without controversy among health authorities except among some as to compulsion 3. There is no practical substitute for fluoridation and water authorities should adopt it where technically and economically feasible in accordance with the relevant legislation
7	Australia (South Aust.)	1964	Select Committee of House of Assembly	1. Fluoride is an aid to reduce dental caries and fluoridation of water supplies is the most convenient cheapest and most effective method 2. Fluoridation is completely safe, has no engineering problems and is a public health measure 3. Fluoridation of the water supplies of the State is desirable
196	U.S.A., Michigan	1964	Select Committee of House of Representatives. 72nd. Legislature	1. The experiment of fluoridation has yet to prove itself and more research is needed 2. 1 ppm of sodium fluoride—a poison— provides no margin of safety 3. No control of fluoride intake into the individual human body is possible 4. Some warning signs are that there are reports of harm from consuming fluoridated water, 170 fluoridated communities have now rejected it and a number of doctors are opposed to fluoridation
1071	U.S.A.	1952	'Delaney Committee' Select Committee appointed by House of Representatives of U.S. Congress to investigate the use of chemicals in foods and cosmetics. 82nd. Congress. 2nd Session. Report No. 2500	1. Fluoridation at 1 ppm presents no hazard to public health according to the major portion of scientific opinion and reduces the prevalence of dental caries in children 2. Fluoridation is mass medication and communities should have the right to say if they will assume the risk inherent in the program 3. The pilot experiments are not completed and in none have the adult population been studied physiologically

APPENDIX 'E'

SUMMARY OF JUDGMENTS REFERRED TO IN THE EVIDENCE IN WHICH QUESTIONS RELATING TO THE FLUORIDATION OF WATER SUPPLIES HAS BEEN CONSIDERED

Ex.	*Country*	*Date*	*Court*	*Parties*	*Summary of Judgment*
17	Eire	1963	Supreme Court Kenny J.	Ryan *v* Attorney-General	1. Fluoridation is not an infringement of any of the fundamental rights guaranteed to individuals under the Eirean Constitution 2. Fluoridation at 1 ppm involves no risk to health or life 3. Fluoridation is not an interference with the rights of an individual or the family 4. Fluoride at 1 ppm will not affect enzymes or damage kidneys or affect their processes and is not connected with goitre. There is no evidence of connection with growth retardation, or periodontal disease, heart conditions or expectation of life 5. Fluoridation is only practicable method to deal with dental caries problem 6. Regular supervision and checks of machinery is a complete safeguard against unreliable machinery
18	Eire	1964	Supreme Court on Appeal from Kenny J.	Ryan *v* Attorney-General	1. Appeal dismissed 2. The minute body changes resulting from ingesting fluoridated water are not a violation of bodily integrity 3. Fluoridation is not mass medication, or a usurpation of parental authority or violation of parental rights
	New Zealand	1964	Privy Council on Appeal from Supreme Court of New Zealand	Attorney-General for New Zealand *v* Lower Hutt Corporation	1. The statutory power to construct waterworks to supply pure water, authorises the Corporation to improve the water quality by addition or subtraction of constituents, and fluoride adds no impurity but improves the water 2. The Judgments in *R. v. Fredericton* (New Brunswick Sup. Ct., Appeal Division) and *Village of Forest Hill v. Municipality of Metropolitan Toronto* (Ontario Ct. of Appeal and Sup. Ct, of Canada) disagreed with so far as not distinguishable 3. Decision of the Supreme Court of New Zealand affirmed
	A stralia (Victoria)	1964	Supreme Court of Victoria	Kelberg *v* City of Sale	1. The City had no statutory authority to fluoridate its water supply 2. A power in a by-law to add any compound of fluorine was *ultra vires*, since some compounds of fluorine were harmful to human beings if consumed
965	U.S.A. Illinois	1964	Supreme Court of Illinois on Appeal from Superior Court of Cook County	Schuringa *v.* City of Chicago	1. Fluoridation at 1 ppm reduces dental decay and does not cause physical harm to the population as a whole, or cause mottled teeth 2. Fluoridation is necessary and suitable for the protection of public health, and is the only practical method of employing the benefits of fluoride 3. Municipalities are entitled to adopt reasonable measures to improve or protect public health, and the rights of the individual must give way to the public good
427	Australia A.C.T.	1964	Supreme Court Bridge J.	Paget & Doughty *v.* Minister of State for the Interior	1. The Minister has statutory authority to fluoridate the Canberra water supply at 1 ppm without contravening a regulation prohibiting the placing of any 'thing' in the water supply, and protecting a public and not a private right 2. Fluoridation is not an offence against the regulation

APPENDIX 'F'

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Fluoridation

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'*Classification and Appraisal of Objections to Fluoridation*' (1960) (K. R. Elwell and K. A. Easlick, eds.) The University of Michigan, U.S.A.

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'*Fluorine Chemistry*' (1964) (J. H. Simons, ed.) Vol. III. 'Biological Effects of Organic Fluorides'. (H. C. Hodge, F. A. Smith, P. C. Chen) Academic Press, New York and London

'*Fluorine Chemistry*' (1965) (J. H. Simons, ed.) Vol. IV. 'Biochemical Effects of Organic Fluorides' (H. C. Hodge and F. A. Smith) Academic Press, New York and London

'*Fluorine Chemistry*' (1964) (J. H. Simons, ed.) Vol. V Academic Press, New York and London

'*Fluorine Intoxication*'—'*A Clinical Hygenic Study*' (1937) K. Roholm, H. K. Lewis & Co. Ltd, London

'*Fluorosis, The Health Aspects of Fluorine Compounds*' (1961) (E. J. Largent, ed.) Ohio State University Press, Columbus

'*Food and Nutrition Notes and Reviews*' Vol. XVIII. Nos. 7 and 8. Pages 49 to 55. July/August 1961

'*The Whalers' Legacy to Cape Barren Island*' (1961) Howeler, Johanna F. (See Coy). Commonwealth Department of Health, Canberra

'*Handbook of Experimental Pharmacology*' (1966) (F. A. Smith, Sub-ed.) Vol. XX. 'Pharmacology of Fluorides', Springer, Verlag, Berlin

'*Mineral Metabolism*' (C. L. Comar and J. F. Bronner, ed.) Vol. II. 'The Elements' (1964) Academic Press, New York

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'*The Toxicology of Fluorine*' (1964) (T. Gordonoff, ed.) Schwabe & Co., Basel

APPENDIX 'G'

The Fluorine Content in Various Foodstuffs

Food	*Fluorine Content (PPM)*
(*a*) *Animal Tissue*	
Cow's Liver, dry wt	5.20- 5.80
Chicken Liver, fresh wt	0.7 – 1.29
Calf Liver, fresh wt	0.2
Cow's Kidney dry weight	6.9 –10.1
Cow's Heart Muscle, dry wt	2.3 – 2.7
(*b*) *Meats*	
Chicken	1.40
Beef	2.00
Round Steak	1.3
Pork	< 0.2
Pork Chops	1.0
Pork Shoulder	1.2
Frankfurters	1.7
Lamb	1.2
Veal	0.9
Mutton	< 0.2
(*c*) *Fish*	
Fish Fillets	1.5
Mackerel	
Boned	< 0.2
With Bones	3.9
Fresh	26.89
Dried	84.47
Canned	12.10
Salmon	
Canned	4.5
Fresh	5.8
Dried	19.3
Sardines	
Canned	7.3
In Olive Oil	16.1
Shrimp	
Canned	4.4
Edible Portion	0.9
Codfish	
Fresh	7.0
Salted	5.0
Oysters	
Fresh	0.7
Crab Meat, Canned	2.0
Herring, Smoked	3.5
Tuna Fish Flakes, Canned	0.1
(*d*) *Eggs*	
Whole	1.2
White	1.5
Yolk	0.6
(*e*) Whole Milk	0.07–0.22
(*f*) Tea	3.2 –178.8
Average of ten samples	97.0
(*g*) *Citrus Fruits*	
Grapefruit	0.36
Edible Portion	0.36
Fresh	0.12
Lemon, Fresh	0.028, 0.051, 0.174
Oranges, Edible Portion	0.34
Oranges, Fruit, Fresh	0.17–0.07
Pomelo, Fruit, Fresh	0.10—0.16

	Fresh Weight	*Dry Weight*
(*h*) *Noncitrus Fruits*		
Apples	0.22 – 1.32	0.13 0.43
Apricot	0.06	0.24
Banana	0.23	0.65
Cherry	0.25	
Cherries, Black	0.18	0.61
Currants	0.12	0.69
Fig	0.21	
Grapes	0.16	
Grape Juice	0.093	
Gooseberries	0.11	0.72
Mango	0.18	
Pawpaw	0.15	
Pear	0.19	
Plum	0.22	0.10
Pineapple	0.14	
Pineapple, Tinned	0.00	
Quince	0.06	0.37
Sweet Melon	0.20	
Strawberry	0.18	
Watermelon	0.11	

APPENDIX 'G'—*continued*

Food	*Fluorine Content (ppm)*	
	Fresh Weight	*Dry Weight*
(i) *Cereals and Ceral Products*		
Corn		
Unspecified	0.62	0.70
Canned	< 0.20	
Yellow	< 0.10	
Germ		8.0 –11.0
Meal, as purchased	0.22	
Flakes		1.33
Ralston		0.58
Wheat		
Whole		0.53
Unspecified	0.7	
Bran	0.29	0.33
Germ A, commercial	1.7	
Germ B, commercial	4.0	
Germ, pure	0.88	1.00
Cream of Wheat		0.55
Flour		
Wheat, White	0.35	
Self-rising	0.45	
Whole Wheat		1.32
White	0.27	0.31
Biscuit		0.0
Baking	0.31	0.35
Bread		
White		0.54
Rice		
Unspecified	0.67	0.76
Whole	< 0.10	
Middle	0.19	
Soybeans		4.00
Buckwheat		
Unspecified		2.00
Whole		1.70
Bran		1.60
Oats		
Unspecified		3.0
Crushed	< 0.20	
Mother's		0.92
Fresh	0.25	0.29
Rye		
Unspecified	0.61	0.69
Black-eyed Peas	0.23	
Cottonseed		
Meal	12.0	
Hulls	12.0–14.0	
Spaghetti		
Canned		1.15
Dry		0.80
Macaroni, dry		0.82
(j) *Vegetables and Tubers*		
Asparagus, Canned		0.48
Beans		
String		0.64
String, Canned		0.67
Green	0.15	1.01
Light Green	0.11	0.73
Lima, Dry		4.51
Lima, Seeds		2.2
Dry		1.04
Dried		< 0.20
Navy, Dry		1.70
Beets		
Unspecified	0.2	
Fresh		0.60
Root		2.8
Leaves, Dry		3.80
Tops		3.4
String	0.32	6.09
Cauliflower		
Fresh		0.45
Flower	0.12	0.86
Leaves	0.08	0.83
Unspecified	1.0	
Cabbage		
Large		9.34
Foreign		15.38
Fresh		0.70
Unspecified	0.13	
Edible Head		3.4
Without Leaves	0.8	9.5

APPENDIX 'G'—*continued*

Food	*Fluorine Content (ppm)*	
	Fresh Weight	*Dry Weight*
Carrots		
Unspecified	0.4	6.92
Fresh		1.30
Root		8.4
Celery		
Unspecified	0.14	
Edible Stalks		8.5
Cress	0.24	4.38
Cucumber	0.20	
Endive	0.2	
Garlic		
Green		17.72
Kale	0.16	
Lettuce		
Loose, Head		11.3
Cabbage	0.30	4.45
Prickly		5.18
Fresh		0.42
Mustard		
Greens	0.15	
Leaves, Salted, Dried		3.0–4.8
Onions		
Green		10.11
Unspecified	0.60	
Parsley		
Tops		11.3
Unspecified	0.8	
Parsnip, Roots		5.5
Peas		
Unspecified	0.6	
Green		6.69
Fresh		0.60
Potatoes		
White		0.96
Unspecified	0.20	
Whole	6.4	22.0
Peelings	0.07	0.35
Irish, Tuber		1.4
Sweet, unpeeled	0.13	
Sweet	< 0.20	
Pumpkin	0.10	
Radish	0.8	
Rhubarb	0.4	
Rutabaga		
Tops		7.0
Roots		2.9
Spinach		
Fresh		1.11
Unspecified	1.8	
Winter	0.44	3.80
Squash, Fresh		0.63
Tomatoes		
Unspecified	0.24	2.40
Fresh		0.53
Turnips		
Greens	0.10	
Tops		1.7
Roots		2.6
Watercress	1.0	
(*k*) *Miscellaneous Substances*		
Peanuts		
Unspecified		1.36
Tops		1.7
Kernel		1.5
Almonds	0.90	0.90
Hazelnut	0.30	0.30
Chestnut		1.45
Shell		0.24
Coconut, Fresh	0.00	
Cocoa	0.5, 0.50	
Plain Chocolate	0.50	
Milk Chocolate	0.50, 1.0	
Molasses	0.00	
Sugar	0.32	
Honey	1.00	
Gelatin	0.00	
Glucose	0.50	
Malt	1.0, 1.5	
Powdered Ginger	1.00	
Baking Powder		
A	220.0	
B	19.0	
C	< 0.1	
Coffee	0.2 – 1.6	
Butter	1.50	
Cheese	1.62	
Pork and Beans, Canned	1.40	

APPENDIX 'G'—*continued*

Food	*Fluorine Content (ppm)*
(*l*) *Wine and Beer*	
Chinese, Shao-shing	0.07
Best Grade	
Second Grade	0.05
Port	0.24
Beer	0.20

From McClure, F. J.: Public Health Rep. 64: 1061, 1949. Prepared by Muhler, J. C. as part of 'Ingestion of Fluoride with Foods' (1967) unpublished contribution to provisional text of a Monograph on 'Fluorides & Human Health' in course of preparation by World Health Organisation.

APPENDIX 'H'

Summary of the Status of Fluoridation in Australia and its Territories

Country	*Communities Fluoridated*	*Population Served*	*Other Particulars*	*Reference*
AUSTRALIA				
Tasmania	3	109,000	Government approves fluoridation by water authorities—Local authorities supply tablets Fluoridation not compulsory and referenda not required	Ex. 532 and sundry exhibits
Victoria	1	3,500	Government attitude undisclosed. Government to introduce legislation to provide that water authorities may fluoridate after referenda	Ex. 1010 Ex. 532
New South Wales	16	3,182,450	Government approves fluoridation—Sydney (Est. pop. 3,000,000) fluoridated 1968. Seven further communities approved, fourteen under consideration	Ex. 532 Ex. 236
Queensland	2	63,150	Government approves fluoridation. Local authorities may fluoridate	Ex. 532
South Australia	Nil	—	No Enabling Legislation. Government attitude undisclosed	Ex. 236
West Australia	—	—	Fluoridation compulsory by legislation 1966	Ex. 532 Ex. 236 Ex. 13
Australian Capital Territory	1	94,000	Commonwealth Government approves fluoridation. Canberra fluoridated (1964)	Ex. 532
New Guinea	1	38,000	Fluoridation compulsory	Ex. 15 Ex. 532

REFERENCES

Ex. 13 Fluoridation of Public Water Supplies Act 1966.
Ex. 532 Flynn, M. J. Schedule of Communities served by fluoridation.
Ex. 1010 Hamer, R. J. Letter to Dr P. R. N. Sutton, 13 Feb., 1968.
Ex. 236 Commonwealth Department of Health: Fluoridation situation in the World. March 1967.
Ex. 15 Fluoridation of Public Water Supplies Ordinance 1965 (Papua and New Guinea).

Summary of the Status of Fluoridation in some Overseas Countries

Country	*Communities Fluoridated*	*Population Served*	*Other Particulars*	*Reference*
Argentina	Nil	—	No established policy by Government. No legislation. Fluoridation planned by Local Authority of Santa Fe (pop. 245,000) to commence 1968 by which 180,000 will receive treated water	Letter Embajada de la Republica Argentina, Canberra. 8 May 1968
Austria	Nil	—	1. No fluoridation because only 50% of population have mains supply, and legal situation does not warrant fluoridation. There are a relatively high amount of underground water supplies. Tablets distributed to pregnant women and babies, and children up to 14 years. Participation is voluntary	Ex. 657 (Note Federal Minister for Foreign Affairs, Vienna. 25 Jan. 1967)
			2. Fluoridated milk distributed in Vienna	Ex. 774 (1963)
Belgium	3	—	Since 1951 Fluoridation operating in triangle between the cities of Namur, Andenne and Marchs-en-Famenne. Natural fluoridation at 1.2 ppm in South West Flanders (pop. 250,000). Authorities and public are not in favour of the extension of experiments	LetterAmbassade de Belgique, Canberra. 26 February 1968
Brazil	58	2,251,000	1. 15 more planned	Ex. 774 (1963)
	86	—	2. Government in favour	Information supplied by Aust. Dental Assn. 10 April 1968
			3. Ministry of Health encourages fluoridation and a Department is responsible for fluoridation of water supplies in towns Passos, Baixo Guandu, Brusque, Palmares and Ribeiras. First fluoridation in 1956. Cities of Rio de Janeiro, Curitiba and other cities fluoridated in State of Rio Grande de Sul. The Department responsible has consulted with the local authorities of the cities, but no referenda have been held	Letter Embassy of Brazil, Canberra. 5 June 1968
Bulgaria	Nil	—	1. Fluoridation at present being examined by the Ministry of Public Health which will make decision. Referenda not required	Letter Consulate General of the People's Republic of Bulgaria, Sydney. 10 April 1968
			2. Fluoriated mineral water available. Two towns planning to introduce fluoridated milk. One town (150,000) planning fluoridation	Ex. 774 (1963)

APPENDIX 'H'—*continued*

Country	*Communities Fluoridated*	*Population Served*	*Other Particulars*	*Reference*
Canada	277	4,960,557	1. Government in favour. Plebiscites required in some Provinces	Infor. ADA. 10 April 1968 and Ex. 657 (Letter Dept. Nat. Health and Welfare, Ottawa—5 Dec. 1967)
			2. Tablets distributed in two Provinces	Ex. 774 (1963)
Ceylon	Nil	—	1. Fluoridation not adopted. Discussion with W.H.O. but no experiments carried out as yet.	Ex. 657 (Letter High Commissioner for Ceylon, Canberra. 27 Feb., 1967)
			2. Proposal to try out pilot scheme in Colombo and adjacent towns but no positive steps taken	
			3. No decision on fluoridation made by Government with whom responsibility lies	Letter High Commissioner for Ceylon, Canberra, 18 April 1968
Chile	61	3,155,103	Government programme of fluoridation first established in 1953. 49.1% of urban population and 29% of total public water supply fluoridated	Letter Consulado-General de Chile, Sydney. 23 May 1968
China	?	—	Canton fluoridated (1965). Government attitude unknown	Ex. 61
Colombia	2	1,000,000	1. Government encourages and compels fluoridation where required in co-ordination with municipal water supply enterprises. No referenda held. Bogota (capital) partially fluoridated and City of Manizales fully fluoridated from 1961. Pilot study in city of Girardot 1953/60—45% decrease in dental caries observed, scheme suspended 1961. 5% of national population fluoridated.	Letter Consulado General de Colombia Sydney. 8 May 1968
			2. Experiments with fluoridated salt	Ex. 774 (1963)
Cuba	—	—	No information but Government in favour	Infor. ADA. 10 April 1968
Czechoslovakia	25	—	1. Scientific Commission of Ministry of Health recommended fluoridation in 1963 proved by examinations in two selected areas indicating 70% reduction in tooth decay. More than 5% of population supplied with fluoridated water. Local authorities decide. No referenda necessary. Fluoridation established 1958.	Letter Consulate General Czechoslovak Socialist Republic, Sydney. 2 April 1968
			2. Fluoride tablets being used experimentally	Ex. 774 (1963)
Denmark	Nil	—	Government attitude to fluoridation unknown. The addition of fluoride to toothpaste is permitted by Government licence, otherwise the addition of fluorine compounds or substances containing fluoride to food and stimulants as well as cosmetic preparations (including agents for the care of teeth and the oral cavity) to increase the fluorine content of the goods is prohibited (excluding aerosol propellants)	Letter Royal Danish Embassy, Canberra. 26 March 1968
El Salvador	1	5,100	Government attitude unknown	Ex. 774 (1963)
Finland	1	46,000	City of Kuopio fluoridated. Generally no need for fluoridation. High natural fluoride content in water (e.g. Hamina 2.6 ppm). Local authorities decide. No referenda necessary	Ex. 657 (Letter Finnish Legation, Canberra. 13 March 1967). Letter Finnish Legation, Canberra. 29 Feb. 1968 and Ex. 774
France	Nil	—	French Government does not allow fluoridation. In 1955 the Conseil Superieur de L'Hygiene decided against fluoridation of water as safety not sufficiently proved. Fluoride should be administered individually on a doctor's or dentist's prescription and controlled by them	Letter Ambassade de France Canberra. 5 March 1968

APPENDIX 'H'—*continued*

Country	*Communities Fluoridated*	*Population Served*	*Other Particulars*	*Reference*
Germany	1	6,000	1. Government does not favour or encourage fluoridation. Favourable consideration will only be given for the execution of tests in specified towns. Tests in Kassel (6,000 pop.) have shown favourable results. Fluoridation first introduced 1952.	Letters Embassy of the Federal Republic of Germany, Canberra. 7/3/68 and 6/5/68
			2. Fluoride is not added for health reasons as well as legal reasons. The West German Water Authority reported against fluoridation 8 July 1966	Ex. 657. Letter Embassy of the Federal Republic of Germany, Canberra. 2/2/1967
Ghana	—	—	Government attitude unknown. Fluoridation not yet investigated in this country	Letter High Commissioner for Republic of Ghana, Canberra. 27/3/68
Great Britan	3	1,400,000	Government in favour. Local authorities decide. No referendum required. Fluoridation begun in 1955. Approved as public health measure 1962. Operating city of Birmingham, County of Anglesey and Borough of Watford. Hoped to commence soon in parts of Lincolnshire, Warwickshire and Borough of Scunthorpe	Letter from British High Commission, Canberra. 15/5/68
Greece	Nil	—	No fluoridation operating. Decision would be made by Government. No referenda necessary. Government attitude unknown	Letter Royal Embassy of Greece, Canberra. 3/4/68
Guatamala	1	420,000	Government in favour. Planned in four communities (pop. 93,838). Fluoridation in Guatamala City commenced 1954, suspended twice for technical and financial reasons. Fluoride tablet experiments planned	Ex. 774 (1963)
Hong Kong	12	3,500,000	Reticulated supply fluoridated	Ex. 774
Hungary	2	—	Government in favour of fluoridation where deemed necessary. Two cities fluoridated (no exact date available). Initiated by Government after consulting scientific circles, but decision by local authority. No referenda required	Letter Consulate General of the Hungarian People's Republic, Sydney. 27/5/68
Iceland	Nil	—	Government not in favour	Letter Consulate General for Iceland, Melbourne. 1/4/68
India	Nil	—	No national policy on fluoridation. Fluoridation not adopted in any form and no experimental work carried out. Water contains natural fluoride above permissible limits. The problem is to de-fluoridate	Letter High Commissioner of India, Canberra. 8/5/68
Ireland	28	1,056,800	Fluoridation compulsory under 'The Health (Fluoridation of Water Supply Act)' 1960. Total population to be ultimately served exceeds 1½ million by 1970. Dublin City, Cork, Limerick, Waterford, Clonmel, Galway, Dundalk and Athlone fluoridated. Technical arrangements for other supplies in hand. Population served by public water supplies is approx. 1¾ million, including approx. 250,000 served by very small supplies which have not been scheduled for fluoridation	Letters Embassy of Ireland, Canberra. 22/3/68 and 4/6/68
Israel	Nil	—	1. Fluoridation not considered necessary but no opposition in principle by the Government. Existing natural fluoride concentration considered adequate in view of diet.	Letter H. I. Shuval, Asst. Professor of Environmental Health Hebrew University Hadassah Medical School, Jerusalem. 6 May, 1968
			2. Tablets to be introduced	Ex. 774 (1963)

APPENDIX 'H'—*continued*

Country	*Communities Fluoridated*	*Population Served*	*Other Particulars*	*Reference*
Italy	Nil	—	1. Government attitude unknown. No fluoridation as most water for domestic consumption contains natural fluoride within safety limits	Ex. 657 Letter Embassy of Italy, Canberra. 22/12/66
			2. No plans to introduce fluoridation in the future	Letter Italian Embassy in Australia. 28/5/68
Japan	—	—	1. Government does not favour or encourage fluoridation. No towns fluoridated	Letter Embassy of Japan, Canberra. 27/5/68
			2. Yamashina (pop. 35,000) experimentally fluoridated 0.6 ppm, 1952-57 when population increase caused additional use of unfluoridated water. Experiment as at 1964 reported by Minoguchi	Ex. 659
			Reference Exhibit 659:	
			Minoguchi, G.—'11th Year of Fluoridation Study at Yamashina in Kyoto and some problem about the fluoridation of water-works in Japan'.	
Liberia	Nil	—	Government favours fluoridation but not needed because of natural fluoride which is assumed to be responsible for good quality teeth of population	Letter Consulate for Liberia, Adelaide. 8/4/68
Luxemburg	Nil	—	Commission established. Fluoridation Plant planned to serve 60% of population	Infor. ADA. 10/4/68
Malaysia	2	163,000	Government encourages States to fluoridate all water supplies. Water supplies in Kluang and Johore Bahru (pop. 163,000) fluoridated. Other supplies in Johore to be fluoridated shortly.	Letter High Commissioner for Malaysia, Canberra. 21/5/68
Malta	—	—	No fluoridation since ground water contains 0.1-0.6 ppm fluoride	Letter Malta High Commission, Canberra. 18/4/68
Mexico	3	284,000	Planned for five communities	Ex. 774 (1963)
Monaco	—	—	Water supplied from France. Government attitude unknown	Letter Consul for Monaco, Melbourne. 10/4/68
Morocco	Nil	—	Planned to introduce fluoride tablets	Ex. 774 (1963)
Netherlands	94	2,277,150	1. Government in favour of fluoridation. Cities fluoridated include Amsterdam and Rotterdam. Further 96 communities to have fluoridation soon (pop. 2,291,500). 156 communities awaiting authorization (pop. 2,272,950). 1/1/68 – 18% of population receiving fluoridated water. Local authorities decide, subject to authorization of Minister for Health and Social Affairs. No referenda	Letters Royal Netherlands Embassy, Australia. 22/12/66 and 10/4/68
			2. Experiments with fluoridated salt	Ex. 774 (1963)
New Zealand	14	1,041,033	1. Government favours and encourages fluoridation. Local authorities decide – referenda not required, but polls have been held. 15 further schemes approved (pop. 164,190) (Feb., 1967). 65% of population who have piped water supply receive fluoridated water.	Letter Director-General of Health New Zealand 12/3/68 and Ex. 657
			2. Rejected by referenda in 11 communities, of which three now receive fluoridated water.	Ex. 657 Letter New Zealand High Commission, Sydney. 3 March. 1967
Norway	Nil	—	1. Fluoridation not legalised. Committee of Ministry of Social Affairs at present studying fluoridation question and whether local authorities should be empowered to legalise introduction of fluoridation	Letter Royal Norwegian Consulate General Sydney 6/2/68
Panama	Nil	—	1. No public water supply fluoridated. (NOTE: This information may not be reliable in view of 2 and 3 below)	Letter Consulado General de Panama, Sydney 29/2/68
	3	373,038	2. One planned	Ex. 774 (1963)
	6	434,999	3. Government in favour	Infor. ADA. 10/4/68 Ex. 77

APPENDIX 'H'—*continued*

Country	*Communities Fluoridated*	*Population Served*	*Other Particulars*	*Reference*
Paraguay	1	201,000	No other information available	Ex. 774 (1963)
Peru	1	—	1. No other information given	Ex. 774 (1963)
	3	100,000	2. Government in favour	Infor. ADA 10/4/68
Philippines	—	—	1. One planned to serve four communities (pop. 3,000,000).	Ex. 774 (1963)
			2. Republic Act No. 3626 provides for fluoridation but not yet implemented	Infor. ADA. 10/4/68
Poland	—	—	Two planned	Ex. 774 (1963)
Portugal	1	10,000	Three years experimental research in one community (pop. 10,000). Reduction in caries evident	Ex. 657 Letter Dr. B. de Pinko, Lisbon. 14/1/67
Puerto Rico	—	—	Reticulated supply fluoridated	Ex. 774 (1963)
Rhodesia	—	—	Government in favour and one scheme approved. Some country districts have excess fluoride	Infor. ADA. 10/4/68
Rumania	1	70,000	Government in favour. Two to three schemes planned	Infor. ADA. 10/4/68 and Ex. 774 (1963)
Singapore	1	—	Island reticulated supply fluoridated. Fluoridation commenced in part in 1956.	Letter Singapore High Commission, Canberra. 23/5/68
South Africa	1	—	Legislation expected to implement recommendations of S.A.R. in 1968.	Letter Sth African Embassy, Canberra. 7/5/68
Sweden	2	126,000	Government permits fluoridation. Permission granted for city of Norrkoping for five years on 16/5/66 (pop. 94,000), Landshrona (pop. 32,000) (1968). Two other cities planning to fluoridate.	Ex. 920 and Letter Royal Swedish Embassy, Canberra. 1/3/68
Switzerland	3	246,000	1. Government encourages fluoridation. Four per cent of population drinking fluoridated water. Aigle (pop. 4,000). Basle (pop. 230,000) and Binningen (pop. 12,000) fluoridated. Local authorities decide usually by referenda. One referendum rejected, Langenthal (pop. 11,000) (15% voted). Fluoridation begun 1960.	Letter Embassy of Switzerland, Canberra. 9/4/68
			2. Fluoridated milk distributed and fluoridated salt available. Tablets available	Ex. 774 (1963)
Syria	Nil	—	Fluoridated milk supplied to private schools. Fluoride tablets distributed	Ex. 774 (1963)
United Arab Republic	Nil	—	1. Government in favour	Infor. ADA. 10/4/68
			2. Two planned	Ex. 774 (1963)
Union of Soviet Socialist Republics	—	—	1. Ministry of Public Health encourages fluoridation. Several towns experimentally fluoridated	Ex. 932 (1960)
			2. Several units artificially fluoridating water and more to be installed.	Ex. 542 (1965)
			3. 60% of cities with low fluorine content to be fluoridated by 1971.	Ex. 544 (1966)
			4. No problem of fluoridation exists in U.S.S.R.	Letter Embassy of U.S.S.R., Canberra. 28/2/68
Venezuela	1	3,500	No other information	Ex. 774 (1963)
Taiwan	1	—	Government in favour. Two planned	Ex. 774 (1963)
Yugoslavia	Nil	—	Local authorities may fluoridate after a proposal by competent Health Centre. Several city councils have considered projects but have never supported them	Letter Embassy of Socialist Federal Republic of Yugoslavia, Canberra. 22/5/68

APPENDIX 'H'—*continued*

Country	*Communities Fluoridated*	*Population Served*	*Other Particulars*	*Reference*
United States of America			1. Federal Government in favour but fluoridation not adopted on a national basis. Local authorities or States decide	Ex. 657 Letter Embassy of U.S.A. 2 March 1967 and sundry exhibits
	3,145	72,329,000 (including 9,902,700 naturally fluoridated)	2. *Compulsory* fluoridation in States of Minnesota, Connecticut and Illinois. Eight of the largest cities in U.S.A. fluoridated, viz., New York, Chicago, Philadelphia, Baltimore, Cleveland, Washington, St Louis, Detroit. Not Fluoridated, Los Angeles, Boston. *Fluoridation possible only after Referenda* in States of Nevada, Maine, Massachusetts, Nebraska, New Hampshire	Fluoridation Census United States Department of Health Education and Welfare 1967 Edit. (Ex. 1075) Ex. 917 and Ex. 1056

REFERENCES

Ex. 657 'Status of Fluoridation around the World' compiled by W. A. Davenport.

Ex. 774 Leatherman, G. H. and Ellis, J. 'Fluoridation Round the World' (1963 edition).

Ex. 61 Taylor, R. L. 'Fluoridation Around the World'.

Ex. 659 Minoguchi, G. '11th Year of Fluoridation Study at Yamashina in Kyoto and some problem about the fluoridation of water-works in Japan'.

Ex. 932 Fletcher, J., Letter. Brit. dent. J.

Ex. 542 Shelepin, A. V., Dr Department of Health, Moscow, Letter to Mr Wright, Adamstown.

Ex. 544 Radio Moscow. Letter to Mr Caldwell, Camden, N. J.

Ex. 917 Rusk, H. A., M.D. Article 'War on Tooth Decay' New York Times 27/8/1967.

Ex. 1056 The American Dental Association—Editorial (February 1968).

NOTE:—This summary is prepared from information available at the time of its preparation.

APPENDIX 'I'

Letter dated April 26, 1957 written by Dr A. L. Russell of the National Institute of Dental Research in U.S.A. to Dr Ionel Rapaport relative to the latter's claim to have demonstrated a parallelism between the prevalence of mongolism and fluoride in the drinking water. (Part I, Ch. 38, paragraph 658 of Report refers).

'Dr Ionel Rapaport
Wisconsin Psychiatric Institute
University of Wisconsin
1552 University Lane
Madison 6, Wisconsin

April 26, 1957

Dear Dr Rapaport,

Please accept my apologies in advance for this very long and detailed reply to your letter of March 26. But analysis of the material you sent me, together with the previous data indicates unequivocably that no association can be demonstrated between fluoride domestic waters and the risk of mongolism, and I feel that you are entitled to the clearest and most complete explanation that I can set down.

To begin with, these are not prevalence data: prevalence must be stated as the ratio of mongol births to all live births, and the only conclusion we can reach from the data is that true prevalence is greater than this material can indicate. Rather, this is a study of *relative risk*, a distinctly different analytic situation. We may take the number of institutionalized cases per 100,000 population as an indicator of risk, if it can be safely assumed that about the same proportion of cases is institutionalized from each area, and if it can be assumed that birth rates for the areas have been relatively uniform over the period of study. (As a matter of fact, other assumptions must be made, such as a uniform life span for mongols from all areas, but these are of lesser importance). You will recall that, accepting these assumptions, I made a preliminary test of relative risk by randomly matching ten fluoride counties against ten non-fluoride counties, an approach which would tend to equalize differences in birth rates and proportions of cases institutionalized. This test indicated that there was no difference in relative risk between the two sets of counties analyzed. This analysis was outlined in detail in my letter of February 8.

Other than philosophical (risk instead of prevalence), there were two principal differences between this analysis and previous ones: (*a*) we had the advantage of voluminous and detailed water histories in the state, so that we were able more accurately to determine fluoride levels in the communities at the several times of pregnancy and birth (obviously of importance in a phenomenon operating from about the seventh to the ninth week of pregnancy), and (*b*) since nearly all of the births in the state, which is about 25 per cent rural, were registered to cities we assumed that a considerable number of births had actually occurred to mothers whose residence during the critical period of pregnancy was in some other community than the place of birth.

For these reasons we offered to furnish data on water histories as required, and asked whether birth data might be rechecked for the mother's usual place of residence. A sample of data so checked was included in your letter of March 18.

Tabulation of these 127 cases indicates that the residence error was rather higher than we had assumed; 58 (45.7 per cent) indicated that the mother lived, during pregnancy, at some place other than the place of delivery. But this was not important in itself; the crucial question involves the amount of distortion the error introduced into the findings for Illinois as published in the Bulletin of the National Academy of Medicine report. The agreement between fluoride in the waters of the city of delivery and in the mother's usual place of residence tabulated out as follows:—

Fluoride in water in city of delivery	Fluoride in water at mother's usual residence			
	Unknown	0.0 to 0.3	0.4 to 0.9	1.0, over
0.1 to 0.3	21%	67%	12%	—
0.4 to 0.9	27%	13%	→60%	—
1.0 and over	17%	20%	4%	→59%

This supplies an estimate of the amount of error introduced into the original table. The estimate for the 0.4 to 0.9 group, however, is based upon too few cases to be very dependable. In addition, there is reason to doubt the fluoride findings for Decatur, which furnished two-thirds of the cases.

(Decatur uses a surface water taken from the Sangamon River, treated for purification, softening, and iron removal. There is reason to doubt that the single estimate of 0.4 ppm F is accurate for Decatur's tap water, since raw surface waters in this part of Illinois rarely contain more than 0.2 ppm of fluorides, and the effect of this treatment would be to reduce slightly the original fluoride content of the raw Sangamon water).

Dropping this category, then, and presuming that the Illinois table would have included only those cases for which usual place of residence had been established, the table would have been altered as follows:—

As presented				*Would have been altered to*			
Population	F	Cases	Rate	Population	F	Cases	Rate
252,246	1.0–2.6	30	11.9	252,246	1.0–1.9	18	7.1
821,287	0.1–0.3	50	6.1	821,287	0.0–0.3	43	5.2
1,073,533		80	7.5	1,073,533		61	5.7

Chi-square value for this altered array is 1,482, indicating that such a difference could be found by accident about once in four instances.

You will notice that the effect has been to reduce the number of high-fluoride cases much more sharply than the number of low-fluoride cases. It is essential to understand that this will tend to be true not only of the Illinois data but of essentially all state-wide data gathered in the United States. This is true because the fluoride water supply is, generally, a very special sort of water supply and the community which employs it is a very special sort of community, plus the fact that such supplies are relatively uncommon. The situation in Illinois is generally true (with a few exceptions) throughout the United States.

APPENDIX 'I'—*continued*

Illinois fluoride water supplies are invariably deep-well supplies, tapping sandstones of the Cambrian or Pre-Cambrian systems at depths of around 1,500 to 2,200 feet. This sort of well is expensive to drill, but furnishes potable water in amounts adequate for cities up to about 50,000 population. Cities of this size may employ such water systems *when available surface or ground water supplies are inadequate.* (These water-bearing sandstones underlie most of Illinois but are utilized mainly in that band of countries across the upper-middle third of the State because of the limited ground-and-surface-water reserves in that area). But the rural family, or the small village near the fluoride city, can usually make do with the scantier flow of water from a shallower (and cheaper) well. Water from such sources is usually low in fluoride (ordinarily up to 0.3 or 0.4 ppm). Hence, when a resident of a nearby area is mistakenly classified as a resident of the fluoride city, the odds are relatively high that the amount of fluoride in the water actually used has been overstated.

Where surface or shallow ground water is abundant, however, the odds are relatively high that a mistake in residence will not result in over or understatement of the amount of fluoride in the water actually used, since the water used in the city and in the surrounding area tends to be much the same in quality.

This introduces a *systematic bias* so that, when this procedure is followed, too many cases will almost invariably be allocated to the high-fluoride classification.

Your analyses, too, are still hampered because you do not have access to detailed water histories. As an example, consider case JDR, born on June 27, 1945, to a 24-year-old mother in Canton. Your birth data check disclosed that her usual place of residence was Cuba, and you have noted a fluoride value of 3.5 ppm for that city. This is the fluoride content of water from a well drilled in 1914 to a depth of 1768 feet into the St. Peter sandstone of the Ordovician system; but since January, 1943 this well has been maintained only for use in an emergency, with the city's domestic water coming from a complex of shallow wells about 35 feet deep. During the critical point in this pregnancy the mother (if she used the city supply) got very little fluoride from water, and the community supply had been low in fluoride for about 18 months. This is a fair example of variation in the fluoride in community supplies, and illustrates how essential detailed and complete information can be in this sort of study. I have spent as long as three weeks in the field on occasion, reconstructing and verifying the fluoride water history of a single city. In some cases such reconstruction is impossible.

So much for the explanation of why your data have behaved in the manner you have observed. A direct analysis of relative risk can be carried out with the data last sent me, if it can be hypothecated that increased risk of a mongol birth is reflected in births of mongols to mothers of younger ages in areas where the risk is greatest. The method of selection of this sample should have no biasing effect upon the ages of the mothers concerned. All of the data (except two cards where information was incomplete) were used. The result, showing average ages with their standard errors, was as follows:—

Fluoride content of domestic water, usual place of residence of mother	*Number of cases*	*Range of Ages*	*Average Age*
Fluoride-free	24	19–44	32.8 ± 1.36
0.1 to 0.3	43	19–44	31.1 ± 1.18
0.4 to 0.9	16	21–43	31.6 ± 1.99
1.0 and over	19	21–43	30.5 ± 1.54
All cases with known fluoride	102	19–44	31.5 ± 0.71
Cases with fluoride content unknown	23	18–40	29.0 ± 1.72

Variance techniques give a more powerful estimate of the differences between the fluoride groups than can be had by consideration of individual standard errors:

Analysis of variance, four groups with known fluoride content of domestic waters

Source of variation	*Degrees of freedom*	*Total squares*	*Mean squares*
Between groups	3	67.2513	22.4171
Within groups	98	5120.2389	52.2473
	101	5187.4902	

$F = 0.429$

which indicates that the differences between these four fluoride groups could occur by pure change about every other time. A scatter-diagram shows no apparent pattern. The coefficient of correlation for the ungrouped data, comparing the age of the individual mother with the fluoride value of the domestic water supply in her usual place of residence, is —0.093 ± 0.0995: in this spot the coefficient would need to be as large as 0.45 or 0.50 before it could be meaningful.

Hence my conviction that no relation with a fluoride domestic water is indicated by these data. This means that our water-history files will not be as useful to you as we had previously supposed. (They remain available to you, of course, for a check of this analysis or any other purpose they may serve).

On the other hand, the analysis has uncovered a slim but potentially promising lead, in the differences between the ages of the mothers in the 'fluoride unknown' group and the larger group for whom fluoride levels could be established. Of the 23 mothers in the 'unknown' group, 5 are 20 years of age or younger; of the 102 mothers in the 'known' group, 3 are 20 years of age or younger. These 'unknown' mothers were, in general, residents of rural areas or communities so small that there was no common water supply. I would predict that they constitute a distinct societal group with characteristics which you can assess better than I; for example, they will probably have incurred such 'childhood' diseases as rubella later in life, and have done more heavy labor, than urban women. They also tend to marry at a younger age and consequently are at risk at a younger age. At any rate, this group stands out on analysis and might repay close study.

While it appears that Dr. Duhl and his group may be more competent to help you in further investigation from the population point of view, I hope that you will continue to call on me or upon our material whenever the occasion suggests.

With best personal wishes, I am

Sincerely yours.

A. L. Russell, D.D.S.

ALR: rg

National Institute of Dental Research'

APPENDIX 'J'

Letter dated January 1957 written by Dr A. L. Russell of the National Institute of Dental Research U.S.A. to Miss J. R. Forrest, Senior Dental Officer, Ministry of Health, U.K. *re* Dr Ionel Rapaport's claim to have demonstrated a parallelism between the prevalence of mongolism and fluoride in the drinking water. (Part I, Ch. 38, paragraph 658 of Report refers).

'Department of HEALTH, EDUCATION, and WELFARE—Public Health Service, National Institutes of Health—Bethesda 14. Md.
NATIONAL INSTITUTE OF ARTHRITIS AND METABOLIC DISEASES
NATIONAL CANCER INSTITUTE
NATIONAL INSTITUTE OF DENTAL RESEARCH
NATIONAL HEART INSTITUTE
NATIONAL INSTITUTE OF MENTAL HEALTH
NATIONAL MICROBIOLOGICAL INSTITUTE
NATIONAL INSTITUTE OF NEUROLOGICAL DISEASES AND BLINDNESS
THE CLINICAL CENTRE
DIVISION OF RESEARCH GRANTS

25 Jan. 1957

Miss J. R. Forrest,
Senior Dental Officer,
Ministry of Health,
Savile Row,
London W. 1., England.

Dear Miss Forrest:

Dr. Ionel Radaport met today with Dr. Arnold, several representatives of the National Institute of Mental Health, and myself.

Previous to today he furnished us with much of his raw data. Good fluoride histories were available to me for the states of South Dakota and Illinois, so particular attention was given to these findings. Two factors were apparent almost at once:

1. The place of birth was used as an indication of fluoride exposed. All of the births in South Dakota occurred in an urban place and all but four of the births in Illinois occurred in an urban place. About two-thirds of the people in South Dakota, and about one-quarter of the people in Illinois, are rural residents.

> (Prior to today Dr. Rapaport had never seen the standard United States birth certificate. It lists (1) place of birth. The entry here is usually the city in which the obstetrical hospital is located, when birth is in a hospital. When birth is in a rural home, the place listed is customarily the town used as the post office address by the family. This should not be confusing, because the second entry (2) describes the 'usual residence of mother', calling specifically for state, county, city or town, and street number, with the instruction to write 'rural' in the *city* or *town* space if the place of residence is outside the city or town limits).

Because of this error (relating his data to place of birth rather than to residence of the mother), it may be presumed that something in the order of three-quarters of his residence data are in error. In this area, for example, about nine-tenths of children who have never lived anywhere except in Prince Georges or Montgomery Counties, Maryland, list their 'place of birth' as Washington, District of Columbia. I know of my own knowledge that such a discrepancy is usually found in South Dakota birth certificates.

2. These data were tied to today's—i.e., current—status of fluoride in water supplies. Two of his South Dakota subjects (ages not known) come from a city, Aberdeen, which had about 2.5 ppm F in its water supply prior to 1938; changed to a surface water running about 0.3 ppm F; and fluoridated to about 1.0 several years ago. Similarly, he thought Maywood to be fluoride-free, though its water carried about 1.4 ppm F up to the time it was connected to the Chicago supply. Ages of his Illinois group averaged over 15 years, ranged up to 56 years; obviously reliable long-term fluoride histories are requisite for such a comparision as he has made.

Of these errors, the first is the more important. It tended to introduce a systematic bias into his array for this reason:

His fluoride-free cities are, generally, large cities like Chicago or Milwaukee. The odds may be estimated as about 99 in 100 that the mother of a child born in Chicago is a resident of Chicago, ranging down to a 50-50 chance that the mother of a child born in Kewanee (F, about 1.00 ppm) was actually a resident of the surrounding rural area. The effect of this bias would, presumably, be such as to produce about his order of results if there were actually no difference between the numbers of patients institutionalized from fluoride and from non-fluoride areas. To test this idea, I assembled the case rates for the following cities, all of which have reliable fluoride histories, all in Illinois: Chicago, Oak Park, Cicero, Waukegan, Quincy, Macomb, Kankakee, Bockford, Elgin, Kewanee, Aurora, Joliet, East Moline, Maywood, Elmhurst, and Galesburg. There was a correlation between fluorides in the water supplied and these case rates; but there was an equally valid inverse correlation between the case rates and the percentage of county population classified as rural. This supported my notion that many of the births attributed to such cities as Aurora were to mothers actually living elsewhere.

Accordingly, I went on to a matched-county study for Illinois, using Rapaport's case data.

Most fluoride-bearing aquifors in Illinois are found in a band extending from east to west across the state, about one-third of the way from the northern toward the southern border, (See: Deatherage, C. F., Klassen, C. W. and Weart, J. G. Fluorides and mottled enamel in Illinois. Illinois Dent J., 8: 194-205, 1939). Since this area is sharply defined I was able to match ten counties with a preponderance of water supplies with 1.5 ppm F or more, against nearby (in five cases, adjacent) counties with no water supply with as much as 0.9 ppm F. This was done without information as to the county location of any of the cities on the list. The urban area in and near Chicago was excluded so that differences in mode of life would be minimized.

I then tabulated the number of institutionalized cases listed as having been born in each of these twenty counties and compared it with the total *county* population, averaged for the years 1930-1940-1950 (to cover the period of the standard deviation of the ages). Population and case rates were as follows:—

	Mean Population 1930-1940-1950	*Number of cases*	*Rate per* 100,000 *population*
Ten Illinois counties with a preponderance of high-fluoride water supplies	536,303	35	6.5 ± 1.1
Ten nearby Illinois counties with no high-fluoride water supplies	335,924	21	6.3 ± 1.4

APPENDIX 'J'—*continued*

This difference could be expected to occurr by chance about five times out of six. The average case rate per county was slightly higher in the low-fluoride counties (6.2 and 6.6 respectively), a difference which, also, might be expected by chance about five times out of six.

This will, I believe, turn out in the end to be the true explanation of Rapaport's data.

Rapaport, himself, is well-meaning but inexperienced. He has agreed to assemble data on the actual place of residence of the mothers of his subjects at the time of pregnancy, and has invited Dr. Duhl of the Mental Institute and myself to check these data against verified fluoride histories, and to visit him in Wisconsin to evaluate his material at first hand. I believe he will do whatever he can to undo the damage his first report may have caused. If my assumptions are correct the final tabulation will resemble the data above.

We will keep you informed of progress along this line, and hope that you will let us know what your own investigations develop. Since I have received essentially the same inquiry from Dr. H. Trendley Dean, a carbon of this letter is being forwarded to him.

With the best of personal wishes, I am

Sincerely yours,

A. L. Russell, D.D.S.

National Institute of Dental Research

CC: Dr. Dean

2/57/236'

D. E. Wilkinson, Government Printer, Tasmania.

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